2601-29

The American Journal of Surgery

EDITOR

ROBERT M. ZOLLINGER, M.D., *Columbus, Ohio*

ADVISORY BOARD

HENRY T. BAHNSON, M.D., *Baltimore;* JOHN M. BEAL, M.D., *New York;* CLAUDE S. BECK, M.D., *Cleveland;* H. GLENN BELL, M.D., *San Francisco;* CLARENCE J. BERNE, M.D., *Los Angeles;* B. MARDEN BLACK, M.D., *Rochester, Minn.;* EDWIN F. CAVE, M.D., *Boston;* CHARLES GARDNER CHILD, III, M.D., *Ann Arbor;* RANDOLPH LEE CLARK, JR., M.D., *Houston;* H. WILLIAM CLATWORTHY, JR., M.D., *Columbus;* FREDERICK A. COLLER, M.D., *Ann Arbor;* HERBERT CONWAY, M.D., *New York;* OSCAR CREECH, JR., M.D., *New Orleans;* GEORGE J. CURRY, M.D., *Flint;* J. ENGLEBERT DUNPHY, M.D., *Portland, Oregon;* DANIEL W. ELLIOTT, M.D., *Columbus;* EDWIN H. ELLISON, M.D., *Milwaukee;* EGBERT H. FELL, M.D., *Chicago;* WILLIAM T. FITTS, JR., M.D., *Philadelphia;* D. P. HALL, M.D., *Louisville;* JAMES D. HARDY, M.D., *Jackson;* J. HARTWELL HARRISON, M.D., *Boston;* STANLEY O. HOERR, M.D., *Cleveland;* CHARLES A. HUFNAGEL, M.D., *Washington;* ARNOLD J. KREMEN, PH.D., *Minneapolis;* WALTER G. MADDOCK, M.D., *Chicago;* K. ALVIN MERENDINO, M.D., *Seattle;* CARL A. MOYER, M.D., *St Louis;* WILLIAM H. MULLER, JR., M.D., *Charlottesville;* D. W. GORDON MURRAY, M.D., *Toronto;* MERLE M. MUSSELMAN, M.D., *Omaha;* SIDNEY W. NELSON, M.D., *Columbus;* THOMAS B. QUIGLEY, M.D., *Boston;* VICTOR RICHARDS, M.D., *San Francisco;* JOHN A. SCHILLING, M.D., *Oklahoma City;* ROBERT TURRELL, M.D., *New York;* JOHN C. ULLERY, M.D., *Columbus;* ROGER D. WILLIAMS, M.D., *Columbus;* HAROLD A. ZINTEL, M.D., *New York*

VOLUME 102

JULY TO DECEMBER

1961

31384

THE AMERICAN JOURNAL OF SURGERY

466 LEXINGTON AVENUE, NEW YORK 17, NEW YORK

MCMLXI

Contents of Volume 102

July 1961

The Eponym Problem	*Claude H. Organ* *Louis A. Sojka*	1
Rhinophyma	*Bruce L. Odou* *Eugene R. Odou*	3
Untimely Lymph Node Biopsy	*Hayes Martin*	17
Pulmonary Embolism. A Clinical and Pathologic Study with Emphasis on the Effect of Prophylactic Therapy with Anticoagulants	*Robert E. Hermann* *John H. Davis* *William D. Holden*	19
Portacaval Shunt: Pre- and Postoperative Management	*M. J. Mackby*	29
Further Observations on the Surgical Treatment of the Dumping Syndrome	*David B. Hinshaw* *Clarence E. Stafford* *Eugene J. Joergenson*	38
Epithelioma of the Tongue. Results of Treatment in 217 Cases	*Harvey Lash* *John B. Erich*	42
Hirschsprung's Disease. A New Modification for Rectosigmoidectomy	*Fayek Ghabrial*	48
Metastatic Carcinoma of the Mandible with Primary Tumor in the Lung	*Jacques E. R. Vrebos* *James K. Masson* *Edgar G. Harrison, Jr.*	52
Traumatic Rupture of the Normal Spleen. Analysis of Eighty-Three Cases	*Timothy Denneby* *Timothy A. Lampbier* *William Wickman* *Ronald Goldberg*	58
Cancer of the Large Bowel in the Young Adult	*Allan Hall* *Robert J. Coffey*	66
Paralysis of the Vocal Cords after Thyroidectomy. A Review	*Matthew H. Evoy*	73
Replacement of Entire Esophagus by Reversed Gastric Tube Procedure (Heimlich-Gavriliu Operation). A Case of Lye Stricture of the Cervical and Thoracic Esophagus	*Boris Schwartz* *Wayne W. Hall*	79
Jejunal Intussusception. An Unusual Complication of the Use of an Intestinal Tube	*Alfred A. Moscarella*	83
Malignant Mixed Tumor of Parotid Gland	*Gerald Fine* *Capt. Richard B. Marshall*	86
Unusual Skin Metastases from Carcinoma of the Gallbladder	*Rustico C. Tongco*	90
Ruptured Pseudocyst of the Pancreas with Associated but Separate Encapsulated Pancreatic Fluid	*Arkell M. Vaughn* *Michael S. White* *John J. Raffensberger*	94
Multiple Primary Carcinoma of the Uterus. Review of the Literature and Report of a Case	*Allan G. W. McLeod* *Denis Cavanagh*	99
Complete Lumbosacral Dislocation. Report of a Case	*Richard K. White*	103
Recurrent Volvulus of the Cecum Associated with Failure of Rotation of the Intestine in an Adult	*Harry V. Armitage*	105

LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

Idiopathic Massive Hemobilia	*L. Kraeer Ferguson* *Moreye Nusbaum*	109
Pseudomembranous Enterocolitis	*Thomas V. Craig*	112
An Investigation of Plastic Dressings for Clinical Use	*Joseph M. Miller* *Deborah A. Jackson* *Charles S. Collier*	115
A Hand Stapler for Use in Radical Mastectomy	*Herbert Volk*	118
The Use of Intramuscular Perphenazine to Control Postoperative Vomiting. A Report of 2,794 Cases	*Berton E. Braly* *Daniel C. Moore*	120
Our Surgical Heritage	*D. P. Hall*	124
Surgery Court Cases	*Howard Newcomb Morse*	128
Book Reviews		130

August 1961

Pacific Coast Surgical Association	*Robert A. Scarborough*	133
Perforated Peptic Ulcer: Results after Simple Surgical Closure	*Newlin Hastings* *Roy Machida*	136
The Natural History and Results of Surgical Therapy for Carcinoma of the Stomach. An Analysis of 250 Cases	*James S. Clarke* *Kenneth Cruze* *Sabri El Farra* *William P. Longmire, Jr.*	143
Acute Massive Hemorrhage from Gastroduodenal Ulceration	*Frank W. Spicer* *John V. Carbone* *Clayton G. Lyon*	153
Treatment of the Massively Bleeding Duodenal Ulcer by Ligation, Pyloroplasty and Vagotomy	*Joseph A. Weinberg*	158
Re-evaluation of Temporary Gastrostomy as a Substitute for Nasogastric Suction	*Gordon Knight Smith* *Jack Matthews Farris*	168
Surgical Treatment of Ulcerative Colitis	*Wiley F. Barker*	176
Gallstones and Diabetes: An Ominous Association	*Fred L. Turrill* *Margaret M. McCarron* *William P. Mikkelsen*	184
Operative Cholangiography	*Richard E. Gardner* *Jack Jew* *Leon Goldman*	191
Trypsin Inhibitor and Proteolytic Enzyme Activity in Dogs after Ethionine-Induced Pancreatitis	*Paul Negulescu* *Harold A. Harper* *Jackson T. Crane* *Leon Goldman*	196
Management of Intestinal Atresia at the Los Angeles Children's Hospital	*William F. Pollock* *William F. Bergin*	202
Carcinoma of the Lung	*Lawrence B. Kiriluk*	217
Regional Blood Flow in Hemorrhagic Shock	*David B. Hinsbaw* *Merle Peterson* *Wilfred M. House* *Clarence E. Stafford* *Eugene J. Joergenson*	224

A Comparison of Methods of Treatment of Extensive Burns at the San Francisco General Hospital	*Albert G. Clark*	231
Experimentation as an Aid in Management of Esophageal Disorders	*Lucius D. Hill* *Edward H. Morgan* *Howard B. Kellogg, Jr.*	240
Experimental Studies in Cryobiology	*Stanley W. Jacob* *Tassadduk H. Mogbul* *Samuel C. Collins* *J. Englebert Dunpby*	254
Surgical Management of Dyskinesia	*Edwin M. Todd* *C. Hunter Shelden* *Robert H. Pudenz* *Benjamin L. Crue, Jr.*	265
Ruptured Aneurysms of the Aortic Sinuses of Valsalva	*Frank Gerbode* *John J. Osborn* *J. Bruce Johnston* *William J. Kerth*	268
Experiences with the Open Correction of Acquired Mitral Valvular Disease with Special Reference to Insufficiency	*K. Alvin Merendino* *David H. Dillard* *Robert A. Bruce* *Leonard A. Cobb* *Arthur M. Anderson*	280
The Role of Pacemakers in the Management of Surgically Induced Complete Heart Block	*William L. Weirich* *Benson B. Roe*	293
Monitors and Clinical Judgment	*Stuart C. Cullen*	300
Basic Problems in the Management of Early Carcinoma of the Cervix Uteri	*Erle Henriksen*	304
Independent Primary Malignant Neoplasms Following Mastectomy for Breast Carcinoma	*Samuel L. Kountz* *William L. Rogers* *Albert C. Daniels*	312
The Treatment of Breast Cancer	*Clare Gray Peterson* *William W. Krippaebne*	321
Urologic Aspects of the Alternating Urachal Sinus	*Frank Hinman, Jr.*	339
A Successful Case of Homotransplantation of the Kidney Between Identical Twins	*Roy Cohn* *Harry Oberhelman, Jr.* *John Young* *Halsted Holman*	344
Book Reviews		351

September 1961

Skill	*Maurice C. Clark*	353
Classification and Prognosis of Thyroid Carcinoma. A Study of 885 Cases Observed in a Thirty Year Period	*Lewis B. Woolner* *Oliver H. Beahrs* *B. Marden Black* *William M. McConahey* *F. Raymond Keating, Jr.*	354

Cervical Lymph Node Metastasis of Thyroid Cancer	*Robert S. Pollack*	388
Management of Gynecomastia	*J. W. Giacobine* *Noel B. Searle*	395
Chondrogenic Tumors of the Jaws	*A. P. Chaudhry* *M. R. Robinovitch* *D. F. Mitchell* *R. A. Vickers*	403
Colonic and Anorectal Function and Disease. Part III	*Robert Turell*	412
Spontaneous Perforation and Rupture of the Esophagus with a Report of Five Cases	*Lew A. Hochberg* *Nicholas Parlamis*	428
Experiences with Management of the Ruptured Appendix	*Capt. Kenneth B. Bonilla* *Lt. Col. Carl W. Hughes* *Col. Warner F. Bowers*	439
The Effect of the Composition of the Ventilating Gas Mixture on Acid-Base Pathways Using the Disc Oxygenator. Studies in Twenty-Nine Patients Undergoing Cardiac Surgery	*George A. Albertal* *Steven Lunzer* *Armand Lefmine* *Dwight E. Harken* *Francis D. Moore* *Ann Mae Fosberg* *Margaret R. Ball*	444
Primary and Secondary Volvulus of the Small Bowel	*Chet R. Lulenski*	453
Malignant Synoviomas. Two Distinct Varieties	*Bholanath Nadkarni* *Robert Brill*	455
Partial Wrist Arthrodesis for Old Ununited Fractures of the Carpal Navicular	*Lt. Comdr. Lawrence H. Gordon* *Don King*	460
Pilonidal Sinus Carcinoma	*John L. Terry* *John C. Gaisford* *Dwight C. Hanna*	465
Internal Drainage of Malignant Pancreatic Cysts as an Effective Palliative Procedure	*Alex G. Shulman* *Richard W. Lippman* *Walter Miller*	470
Intestinal Obstruction Caused by Persistence of the Umbilical Vein as a Transperitoneal Structure	*Jessie L. Ternberg* *Kathleen Winters*	473
Choledocholithiasis and Common Bile Duct Obstruction	*William G. Pace*	475
A Double Lumen Nasogastric Tube in Gastric Surgery	*John O'Leary Nolan*	478
A Versatile Surgical Forceps	*John L. Keeley* *Arne E. Schairer*	480
Diagnostic Corner	*Sidney W. Nelson*	482
Our Surgical Heritage	*D. P. Hall*	486
Book Reviews		491

October 1961

Treatment of Pancreatitis	*Thomas T. White*	493
Thyroglossal Duct Cysts and Sinuses: Results of Radical (Sistrunk) Operation	*Paul M. Brown*, *Edward S. Judd*	494
Aneurysms of the Sinus of Valsalva. Three Different Clinical Pictures	*James W. Pate*	502
A Comparative Evaluation of Prosthetic and Homologous Artery Grafts Used in the Treatment of Occlusive Disease of the Superficial Femoral Artery	*W. Graham Knox*	507
Experimental Appraisal of the Agents Employed as Angiocardiographic and Aortographic Contrast Media. IV. Physical and Chemical Properties	*Duncan A. Killen*, *Edward M. Lance*	512
Intermittent Peritoneal Lavage	*J. J. McCaughan, Jr.*, *Curtis McGown*	519
Extradural Hemorrhage in the Posterior Fossa. Diagnosis and Treatment with a Report of Two Surgically Treated Patients	*John M. Meredith*	524
The Use of Full Thickness Grafts and Suction Following Radical Mastectomy	*Burt B. Smith*	532
The Surgical Treatment of Radiation Dermatitis Following Irradiation of Benign Diseases	*H. P. Royster*, *H. B. Lebr*	535
Incompatible Blood Transfusion and Lower Nephron Nephrosis	*Mussarat S. Khawaja*, *Thomas G. Samter*, *Harris B. Shumacker, Jr.*, *Edward Joe Armbruster*, *Jun-ichi Shikata*	542
Experiences with Large Infusions of Gamma Globulin	*W. W. Coon*, *Vivian Iob*, *E. F. Wolfman, Jr.*, *P. E. Hodgson*, *Madeline McMath*	548
Treatment of Chronic Nonspecific Ulcers of the Leg with Adenosine-5-Monophosphate. A Controlled Study	*Charles V. Menendez*, *Oscar Creech, Jr.*	554
A Study of the Lethal Mechanism in Experimental Closed Loop Strangulated Obstruction of the Small Intestine	*M. D. Turner*, *J. B. Grogan*, *G. W. Truett*	560
A Severe Wringer Injury with Osseous Involvement	*H. William Porterfield*, *Robert E. Morrow*, *Harold M. Trusler*	569
Diagnosis of Torn Meniscus	*Gerald Martin*, *Arthur C. Connor*	573
Improved Simple Manual Dermatone	*John T. Bate*	576
A New Diagnostic Sigmoidoscope for General Use	*Richard S. Wilson*, *Furman T. Wallace*	578
Depth Perception in Proctoscopy	*Robert B. Lewy*	581

Extraperitoneal Thoracic Aorta to Femoral Bypass Graft as Replacement for an Infected Aortic Bifurcation Prosthesis	*F. William Blaisdell* *Gaetano A. DeMattei* *Peter J. Gauder*	583
Spontaneous Perinephric Hematoma Secondary to Renal Tumor	*David Presman* *Donald Rolnick* *Fred Portney*	586
Innominate Artery Loop as a Pulsating Mass in the Neck	*Robb H. Rutledge*	594
Gastrocolic Fistula Secondary to Carcinoma of the Stomach. Management of an Unusual Case Surviving Six Years after Radical Operation	*Joseph L. Ponka* *Robert H. Rice*	596
The Air-Sucker's Gastric Rupture Syndrome	*Viggo B. Olsen* *Thomas J. Foley*	602
Kultschitzky-Cell Carcinoma (Carcinoid) of the Appendix with Metastasis	*W. D. Latham* *H. S. Arnold* *S. Ede*	607
Surgery Court Cases	*Howard Newcomb Morse*	611
Our Surgical Heritage	*D. P. Hall*	614

November 1961

Relaxation and Alertness	*Maurice C. Clark*	619
Pathologic Study of 217 Cases of Epithelioma of the Tongue	*Harvey Lash* *John B. Erich* *Malcolm B. Dockerty*	620
Shoulder and Elbow Lesions of Baseball Players	*I. C. Middleman*	627
Female Urinary Stress Incontinence	*Herbert F. Newman* *Jane D. Northup*	633
Serum Lactic Dehydrogenase Activity in Acute Abdominal Disease	*Robert M. Goldwyn* *Robert H. Resnick* *Walter P. Ellerbeck*	643
Intra-arterial Chemotherapy for Metastatic Cancer to the Brain	*Irving M. Ariel*	647
Bilateral Adrenalectomy for Advanced Breast Cancer. Review of the Literature and Analysis of Twenty-Five Cases	*Angelos A. Kambouris* *Harry C. Saltzstein* *Schayel Scheinberg*	651
Observations on Coronary Sinus Flow	*John G. Pantzer* *Harris B. Shumacker, Jr.*	657
Chronic Occlusive Disease of the Carotid Arteries with Nondiagnostic or Misleading Pressures on the Retinal Arteries	*Maurice C. Smith* *William F. Hoyt*	661
Assessment of the Patency of the Peripheral Arterial Tree by Electronic Pulse Recordings	*Joseph E. Armbruster*	668
Prophylactic Use of Procaine Amide (Pronestyl) in Ventricular Fibrillation During the Production of Intracardiac Defects in Dogs under Hypothermia	*F. L. Raffucci* *Marino Blasini* *J. R. Vilaró* *A. Fernández*	677

Types of Renal Hypertension Correctible by Surgical Therapy. Correlation of Renal Arteriography and Pathology	*James M. Stokes*	680
Replacement of the Cervical Segment of the Esophagus with an Aortic Homograft	*Glenn A. Young* *Angelo E. Dagradi*	687
Acute Superficial Saphenous Thrombophlebitis	*Jessie L. Ternberg* *Porter M. Bailes Jr.* *Harvey R. Butcher, Jr.*	691
Surgery for Gallbladder Disease in Ohio. A Survey of 3,085 Operations	*E. C. Weckesser*	695
Intrauterine Torsion of the Spermatic Cord	*Robert H. Whittlesey*	699
The Use of an Internal Polyethylene Shunt During the Resection of a Carotid Aneurysm	*B. Eiseman* *Bruce C. Paton* *Howard Hogsbead*	702
Experimental Study of Polyester Fiber Suture	*R. W. Postlethwait* *M. L. Dillon* *J. W. Reeves*	706
Surgical Management of a Recurrent Solitary Multilocular Nonparasitic Cyst of the Liver	*John P. Minton* *David L. Kinsey*	710
Actinomycosis of the Sigmoid Colon	*Pierce W. Powers* *Lt. Comdr. Scott G. Kramer* *William L. Drake, Jr.*	713
Torsion of the Normal Ovary	*Robert L. Berger* *George Robbins*	716
Reconstruction of a Large Defect on the Entire Thickness of the Chest Wall	*George Warren Pierce* *Thomas Wiper* *George Magladry* *E. Horace Klabunde* *Vincent R. Pennisi* *Robert Fagella*	720
Tumors of Major Peripheral Nerves	*John J. Byrne* *John M. Cahill*	724
The Stomped Pancreas Death	*Robert W. Huntington, Jr.* *James Colt* *Fred Owen* *Milton Younger*	728
Malpractice: A Primer for the Medical Defendant	*Miley B. Wesson*	732
Our Surgical Heritage	*D. P. Hall*	739
Book Reviews		743

December 1961

Papers of the Society of Head and Neck Surgeons

These papers were presented at the seventh annual meeting held on March 15–17, 1961 in Miami Beach, Florida

Presidential Address. Seventh Annual Meeting	*Arnold J. Kremen*	745
A Study of Thyroid Cancer in Children	*Theodore Winship* *Randi V. Rosvoll*	747

Median Labiomandibular Glossotomy. Trotter's Median (Anterior) Translingual Pharyngotomy	*Hayes Martin* *H. Randall Tollefsen* *Frank P. Gerold*	753
Parotidectomy in the Treatment of Chronic Sialadenitis	*Oliver H. Beahrs* *Kenneth D. Devine* *Lewis B. Woolner*	760
Acinic Cell Carcinoma of the Parotid Gland. A Clinicopathologic Review of Eleven Cases	*Theodor B. Grace* *Paul H. Lober* *Stuart W. Arbelger*	765
Parotidectomy for Inflammatory Lesions	*Samuel L. Perzik*	769
Auriculotemporal Syndrome (Frey's Syndrome) Following Surgery of Parotid Tumors	*H. Mason Morfit* *David Kramish*	777
Statistical Evaluation of 115 Cases of Carcinoma of the Epiglottis (1943 to 1952)	*Darius K. Shahrokh* *Kenneth D. Devine* *Edgar G. Harrison*	781
Total Laryngectomy and Cervical Esophagectomy. Reconstruction Employing a Greater Curvature Gastric Tube	*John C. Hawk, Jr.*	789
Treatment of Advanced Tongue Cancer by Suprahyoid Total Glossectomy and Excision of Floor of the Mouth	*Marcel Dargent*	793
Cavernous Hemangioma of the Oral and Hypopharynges	*Edwin W. Cocke, Jr.*	798
Cancer of the Palate. Plastic Repair Versus Prosthesis	*M. T. Edgerton* *R. V. DeVito*	803
Bilateral Radical Neck Dissection Performed in Stages. Experience with 467 Patients	*Edgar L. Frazell* *Oliver S. Moore*	809
A Muscle Cover for the Carotid Artery After Radical Neck Dissection	*Charles J. Staley*	815
Results of Cytologic Studies of Wound Washings and Use of a Local Cytotoxic Agent in Head and Neck Surgery	*Kumao Sako* *Frank C. Marchetta* *Jorge Badillo* *Eugene Burke*	818
Squamous Carcinoma of the Oral Cavity and Throat and Its Clinicopathologic Correlation	*Russel P. Sherwin* *Anthony Zovickian*	823
Palliation Achieved in Carcinoma of the Head and Neck with Intra-Arterial Chemotherapy	*Calvin T. Klopp* *David Forsythe Smith* *T. Crandall Alford*	830
Fibrous Dysplasia of the Facial Bones	*Clifford L. Kiehn* *John D. DesPrez* *Alvin H. Harris*	835
A Simplified Technic for Regional Perfusion. Report of Its Use in a Series of Twenty-three Cases of Malignancy of the Head and Neck	*Frederick M. Golomb* *Alden B. Hall* *Kenneth R. Cox* *Charles F. Schetlin* *Stephen L. Gumport* *Jane C. Wright*	839

Comparative Incidence of Stones in the Common Duct in Acute and Chronic Cholecystitis (and Their Relationship to the Number of Calculi in the Gallbladder)	*Alex E. Pearce*	847
Coccygectomy	*Kenneth F. Spence, Jr.*	850
Rotation of Esophageal and Pharyngeal Flaps for Reconstruction of the Cervical Esophagus	*Frank C. Marchetta* *Kumao Sako* *Patrick J. Creedon*	854
Congenital Aneurysm of the Jejunum	*Henry Hug* *Charles D. Branch*	859
Multiple Leiomyosarcomas of the Jejunum Metastasis to Inguinal Lymph Nodes	*T. K. Das Gupta* *Leonard H. Kolb* *John C. Grubn*	861
Papilloma of the Ampulla of Vater	*Marion C. Anderson* *Wesley H. Gregor*	865
Surgery Court Cases	*Howard Newcomb Morse*	872
Our Surgical Heritage	*D. P. Hall*	874
Author Index		879
Subject Index		881

COPYRIGHT, 1961
BY THE REUBEN H. DONNELLEY CORPORATION
All Rights Reserved

Printed in the United States of America

The American Journal of Surgery

VOLUME 102 JULY 1961 NUMBER ONE

EDITORIAL

The Eponym Problem

Only recently a fifty year old man came to me with a recurrent hernia which had been repaired on two previous occasions. In reviewing the two reports on these operations, such terms as Hesselbach's triangle, Scarpa's fascia, pampiniform plexus and the ligaments of Poupart, Cooper, Henle and Hueck were present. Scarcely anyone save an eponym enthusiast would have known what type of repair had been performed at these two operations.

Eponyms have long been a source of confusion to both anatomist and surgeon. These designations appeared early in the history of both sciences and in the past fifty years one eponym has come to be compounded on another. The lack of universal agreement among anatomists on terminology, along with a degree of egotism, has almost precluded the elimination of these nondescriptive surnames in the surgical sciences. This fad has progressed to the stage where the slogan now is, "If in doubt, just say Morgagni."

In the ten year span from 1944 to 1954, 618 articles appeared in the literature on nomenclature, of which only twelve pertained to anatomic terminology. Seven of this group were on the bronchopulmonary segments. A much needed clarification of anatomic terms took a rear seat during these years while preferential treatment was given to outlining terminology on "disorders of menstruation" and "African mollusks." It must be pointed out that this problem is not peculiar to gross anatomy and surgery for eponyms are employed with perhaps even greater frequency in neuroanatomy, histology, pathology and clinical medicine. In a current textbook of gross anatomy [1] 210 eponyms are used and over one hundred such terms appear in a popular surgical anatomy reference [2].

The initial effort to standardize anatomic terminology dates back to *Basle Nomina Anatomica* (*B.N.A.*) in 1895. In 1933 the Anatomical Society of Great Britain and Ireland published a revision of the *B.N.A.* (*B.R.*); at approximately the same time the Germans published a revision of the *B.N.A.* entitled: *Jena Nomina Anatomica* (*J.N.A.*). The fourth International Congress of Anatomists, held in Milan in 1936, set up an International Anatomical Committee, but little progress was made before the outbreak of World War II in 1939. The evolution of differences in terminology since that date has produced *Nomina Anatomica* (*N.A.*) [3], which was approved by the International Nomenclature Committee (appointed by the Fifth International Congress) in Paris in July, 1955. There was unanimous adoption of the following principles: (1) Each structure, with few exceptions, shall be designated by one term only; (2) every term in the official list shall be in Latin; (3) each term shall be short and simple; (4) terms shall be primarily memory terms, preferably informative and descriptive; (5) structures closely related topographically shall have similar names as far as possible; (6) differentiating adjectives shall, in general, be arranged as opposites; (7) *eponyms shall not be used in the official nomenclature of gross or macroscopic anatomy.*

The disadvantages of eponyms are as follows: (1) Multiple eponyms have been attached to the same structures. The parathyroids have been referred to as Gley's or Sandström's

LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

glands. The ileocecal valve has been alluded to as the valve of Varolius or as Bauhin's valve. (2) This kind of designation is of no descriptive value. Such terms as Home's lobe, Cruveilhier's plexus and Broca's area give no clue as to whether veins, arteries, nerves or lymphatics are under discussion. (3) Eponyms serve as a source of error when they do not describe the structure accurately. The ligament of Treitz is substituted for the suspensory muscle of the duodenum; Gerdy's hyoid fascia for the superior carotid triangle. (4) The same surname has been used to designate several entities. When the term, Cooper's ligament, is used, one is not sure as to which Cooper's ligament is being referred to. Is it the ligament in the inguinal canal or at the elbow? Or would, by chance, the suspensory ligaments of Cooper of the breast be meant? Another example is the gland of Cloquet. Does this mean the femoral canal lymph node or the detached forepart of the lacrimal gland? (5) Historical inaccuracies continue to be perpetrated by eponyms. The origin and description of Hesselbach's triangle and Poupart's ligament illustrate this point [4]. (6) Eponyms have come to be compounded upon each other. For example, Rolando's points are located at the upper and lower end of the fissure of Rolando. Bartholin's duct and canal are anatomically located with reference to Bartholin's gland. (7) Pathologic entities are sometimes confused with anatomic data. (8) Eponyms are needlessly redundant, as the long thoracic nerve of Bell or the subareolar plexus of Sappey illustrate.

To retain eponyms because they are established or to honor men who have labored in these fields seems untenable. MacDermot [5] has reminded us that, to the contrary, the giants in the field of anatomy like Morton, Jenner, Harvey and Vesalius have not been so honored.

Departments of anatomy in most medical schools have been endeavoring to teach basic terminology and eliminate eponyms for a number of years, and with the issuance of *Nomina Anatomica* have received additional impetus. It is now that a concerted effort must be made, particularly in the surgical sciences, to use *Nomina Anatomica* in both teaching and writing so that anatomic terminology can be properly correlated with the teaching of gross anatomy in the preclinical years. This would also be in keeping with the trend in recent years to place the surgical sciences on a more basic scientific footing. Following is a list of some of the most commonly used eponyms with the corresponding correct anatomic term.

Eponym	Corrected Term
1. Ampulla of Vater	Ampulla of common duct
2. Alcock's canal	Pudendal canal
3. Bochdalek's foramen	Pleuroperitoneal foramen
4. Burns' space	Suprasternal space
5. Chassaignac's tubercle	Carotid tubercle
6. Camper's fascia	Superficial layer of superficial fascia
7. Colles' fascia	Deep layer of superficial perineal fascia
8. Cooper's ligament	Pectineal ligament
9. Duct of Santorini	Accessory pancreatic duct
10. Duct of Wirsung	Main pancreatic duct
11. Foramen of Monro	Interventricular foramen
12. Bartholin's gland	Greater vestibular gland
13. Gland of Cloquet	Lymph node of femoral canal
14. Hunter's canal	Adductor canal
15. Ligament of Treitz	Suspensory muscle of duodenum
16. Petit's triangle	Inferior lumbar triangle
17. Pampiniform plexus	Spermatic plexus
18. Poupart's ligament (Vesalius', also)	Inguinal ligament
19. Spigelius' line	Semilunar line
20. Stensen's duct	Parotid duct
21. Space of Retzius	Prevesical space
22. Sphincter of Oddi	Sphincter of common duct
23. Tail of Spence	Axillary tail of breast
24. Vein of Mayo	Prepyloric vein
25. Wharton's duct	Submaxillary duct

REFERENCES

1. Morris' Human Anatomy, 11th ed. Edited by Schaeffer, J. P. New York, 1953. Blakiston-McGraw.
2. Anson, B. J. and Maddock, W. G. Callender's Surgical Anatomy. Philadelphia, 1952. W. B. Saunders Co.
3. Nomina Anatomica. Revised by The International Anatomical Nomenclature Committee. Baltimore, 1956. Williams & Wilkins Co.
4. Keynes, M. Battle of eponym. *Brit. M. J.*, 1: 96, 1954.
5. MacDermot, H. E. The problem of the eponym. *Canad. M. A. J.*, 53: 74, 1945.

Claude H. Organ, Jr., M.D.
Louis A. Sojka, M.D.
Department of Surgery
Creighton University School of Medicine
Omaha, Nebraska

Rhinophyma

BRUCE L. ODOU, M.D. AND EUGENE R. ODOU, M.D., *Montebello, California*

ALMOST every large city has one or more "whiskey noses" walking the streets. (Table I.) We believe that many of these people go through life ignorant of the existence of adequate treatment. Not only are patients unaware of the possibility of treatment, but many physicians share this idea concerning this terrible affliction. It is the duty of every doctor to inform these "repulsives" of results obtainable with modern day therapy. Most of these patients come to the physician for treatment of other conditions, completely unaware that they can be cured. With our attention focused on cancer, heart disease, etc., we easily overlook the other fields of medicine and possible help for our patients. It is with this intent this paper is being offered. When we take into consideration the total number of patients and those which are operated upon, the percentage of the latter is not as large as expected.

In reviewing the literature of over 150 articles on rhinophyma in the English language and of the better known foreign authors, there are only 103 proved cases of rhinophyma. Our criterion for a proved case of rhinophyma was based on its representation as authentic in a photograph or drawing. Surprisingly, the literature contained several repeats or photographs written up as if they were "new" cases. We

TABLE I
SYNONYMS OF THE MORE EXPRESSIVE TERMS USED FOR RHINOPHYMA

Whisky nose	Bulbous nose
Acne hyperplastica	Potato nose
Rum nose	Lymphangioma
Hammer nose	Toper nose
Double nose	Rum blossom
Fatty nose	Brandy nose
Growing nose	Grog blossom
Fibroma molluscum	Wine nose
Cystadenofibroma of nose	Hypertrophic rosacea
Jug nose	*Couperose, copperas* (French)
Monstrous hypertrophy of nose	*Kupefernase* (German)
Copper nose	*Pfundnase* (German pound nose)
Nodular nose	*Elephantiasis des buveurs*
Elephantiasis of nose	Rummy nose
Pound nose	Strawberry nose
Pachydermatosis	
Pseudoelephantiasis of nose	

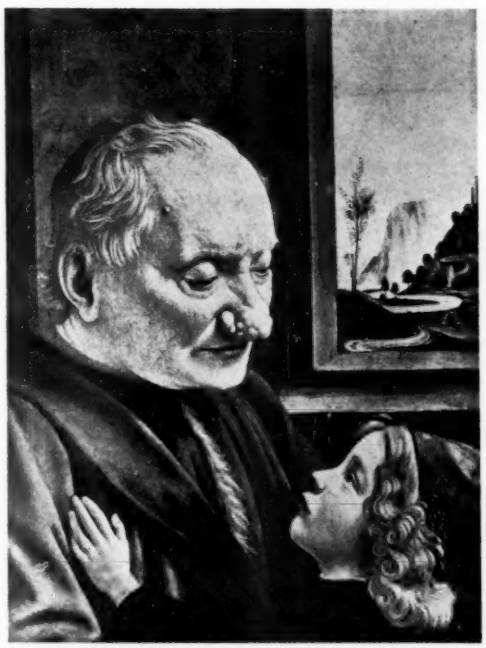


FIG. 1. "An Old Man and His Grandson" by Domenico Ghirlandaio, 1449–1494. Rhinophyma?

FIG. 2. "Unknown Sitter" by a Holland master, in museum in Stockholm.

occasionally had the impression that authors were carried away when they discussed the many patients they had operated upon but were somehow reticent to show photographs of "these numerous cases." However, assuming they did have the number of cases claimed and taking into account surveys made of this condition in Boston, Philadelphia and Los Angeles by various authors there were a total of 253 to 258 cases found in the entire medical literature.

History shows no records or descriptions of this condition much before 2,000 years ago. The Bible makes no mention of this ugly disease. We have recently consulted several authorities in the Biblical field and they could find no such condition. It is our opinion the disease was certainly present during this time, and it must have been confused with leprosy or tuberculosis. In modern times these patients are still often cast aside and, certainly, this did occur more frequently in Biblical times.

Phillip K. Allen in a discussion at the conclusion of Crabtee's article [*21*] states it was known to Hippocrates and was described by the early Arabians. We have not been able to find the source for his statement.

Artists next fill in the historical background of this disease. Hanging in the Louvre is a picture, "An Old Man and his Grandson" by Domenico Ghirlandaio (1449–1494), a famous Florentine painter. (Fig. 1.) This clearly shows

FIG. 3. Large Nose Doctor (late 1600). "For healthy people, I am a doctor. God help the sick. My large headgear embraces profound and numerous thoughts. My costume connotes the Art that I possess. What may be concealed in the urine my long nose detects." (From Hollaender's *Caricature and Satire in Medicine*, 1930.)

rhinophyma although some say it is representative of metastatic disease as well.

Another equally famous painting of rhinophyma is one which hangs in the Prado in Madrid, "Dance of Death" by Holbein the Younger (1497–1533). These and many other paintings and drawings relating to rhinophyma may be seen in Dr. Eugene Hollaender's books, *Medicine in Classical Arts* and *Caricature and Satire in Medicine* [*62*]. (Figs. 2, 3, 4 and 5.)

According to Joseph [*70*], the first rhinophyma was operated upon in 1629 by Daniel Sennert. Following this confirmed surgery, there are few mentions of rhinophyma until the last half of the nineteenth century when numerous surgical and medical cures were reported. It was during this time that all the modern cures of this disease were tried; how-

FIG. 4. From a seventeenth century pamphlet. (From Hollaender, 1930.)

ever, we see recent articles on discovering "new" cures or technics.

In Shakespeare's *King Henry V*, Fluellen's allusion to Bardolph's countenance aptly expresses the true appearance of rosacea: "His face is all bubbles, and whelks, and knobs, and flames of fire; and his lip plows at his nose, and it is like a coal of fire, sometimes blue and sometimes red."

Another play popularizing probable rhinophyma was *Cyrano de Bergerac*. Edmond Rostand, the author, was but twenty-nine years of age when his play opened at the Theatre de la Porte Saint-Martin on the night of December 28, 1897 and swiftly took the world by storm. No other play in history, before or since, has ever attained a popular success so instantaneous or so enormous.

Other successes have also occurred in connection with this usually terrible affliction. A recent Hollywood character made millions on the basis of his reputed alcoholism, distaste for water and his large red nose.

In reviewing the literature, many interesting observations are made. Coldrey [*20*] related that his patient refused surgery for some time because a friend had informed him that if anything was done he would bleed to death. Gottheil [*54*] stated it was essentially a European disease, being most prevalent in Germany, France, etc., the so-called "wine countries." Ohmann-Dumesnil referred to "stiff drinkers." He also noticed a peculiarity in that they are generally averse to any form of treatment.

The youngest case reported is of a patient, twenty-one years of age at the time of surgery [*119*]. There were two other patients with the affliction who were definitely in their

FIG. 5. Gerhard Janssen, a master glass etcher. Born in Holland and painted by U. D. Metzger in 1725. The legend just above his head reads, "Nasutus sed acutus" (large nose but wise), and the sentence in the frame reads, "A misshapen nose indeed, but a man of talent and wisdom. Art, Work, and Industry bring praise and reward." (From Hollaender, 1930.)

FIG. 6. Marked cartilage deformity produced by rhinophyma. (Courtesy of Dr. Rees.)

twenties. No cases were reported of sufferers from this disease as being in their thirties. Only three others were reported below the age of forty-five years. Thus, the disease is predominantly one occurring in men over the age of forty-five.

UNUSUAL CASES

"There is a little story told here, that a while before he gave up all active employment and quit mixing with his fellows he was behind a counter in a grocery store, where he was working temporarily; and a little boy who had never seen him came in to buy a nickel's worth of peanuts. As the boy walked up to the counter, not seeing the man's face, which was turned the other way just at that time, he said, 'Mister, give me a nickel's worth of . . . .' Then, suddenly getting a sight of the man's nose, he started for the door, saying, 'Oh, Good God, what a nose! and never stopped to buy any peanuts at that store." [*109*.]

Rhinophyma may be markedly deforming. Dr. Rees (Fig. 6) shows a most interesting case demonstrating the deforming nature of this hypertrophy upon the cartilaginous structures of the nose. Upon removal of this growth the nose assumed its normal shape as the cartilages were pushed aside and not destroyed.

Cinelli's case [*18*] is extremely interesting in that it describes a seventy-seven year old male who had a twenty year history of difficulty in breathing due to the obstruction of his airway and who finally came to see a doctor because of this condition.

The first actual photograph of this condition is an excellent one taken by Duhring [*30*] in 1871. The accompanying text read: "The body of the nose at its greatest circumference measures 6.5 inches. When he shakes his head the whole nose moves as a pendulous tumor and he complains of its weight; beyond this, but little inconvenience is experienced . . . considering the ageing condition of our patient, it was not deemed expedient to resort to any operation, he himself preferring to keep the nose as it was the few years he might have to live."

Another excellent article to be found in the older literature is by Squire [*135*] in 1889 and is accompanied by a beautiful colored drawing.

Threatened suicide has been described. Crabtree [*21*], for one, had such a patient, a woman whose rhinophyma produced extreme personal embarrassment.

"Victims of this trouble suffer great humiliation, unemployment and may even become social recluses" [*109*].

"Mentophyma" was first described in 1932. This, of course, was pure hypertrophy of the chin by rhinophymatous changes. Sams [*119*] reported rhinophyma with "mentophyma" in a woman in 1932. Rees [*113*] has described this condition in association with the ear. Perhaps this should be called "oto- or pinnaphyma."

"The weight of the tumor was such that it produced stenosis of the nostrils and he had to breathe through his mouth, a condition, which although uncomfortable at first, finally became intolerable."

Numerous cases have been recorded in which the growth of the nose has to be held aside when the patient partook of food or drink [*146*]. (Fig. 7.)

ETIOLOGY, PATHOGENESIS AND PATHOLOGY

Rhinophyma is probably the final stage of acne rosacea with marked hyperplasia of the sebaceous and connective tissue elements. Virchow, in 1846, was the first person to link rhinophyma with acne rosacea. To this day

FIG. 7. Whitham's case—severe rhinophyma. We consider this case to be one of the most severe distortions shown in the world's literature. The results of shaving were excellent in the hands of Dr. Whitham [*153*].

FIG. 8. Chief Wa-Ha-Gun-Ta (American name is Captain John Smith) of the Chippewas. Age, 127 years; and, at the time the photograph was taken, still active and hunting in Glacier National Park [*124*].

this is the general opinion of physicians who have studied this disease. This hypertrophy occurs over a period of years producing lesions ranging from the size of a comedo to a large bulbous, nodular, discolored and lobulated mass. Pitted scars, depressions and large follicular openings may be seen. We have observed the characteristic sebaceous cystlike odor coming from this disease and frequently the patient also complains of this odor. The end result of rhinophyma is a large, deep cherry red nose of grotesque appearance. Pressure over the growth produces the characteristic coil of a sebum and keratin substance.

Vidal and Leloir have distinguished two forms according to the histopathologic findings. (1) glandular form (acne hypertrophica) which is most often due to the abnormal size and number of sebaceous glands, and (2) fibrotelangiectatic form in which the proliferation of the connective tissue is predominant. Between these two extremes intermediate varieties can be observed.

The sequel to acne rosacea is accepted as fact on the basis of similarity of the pathologic pictures of the two diseases. Acne rosacea is a subacute or chronic congestive disease of the nose and fleshy areas of the face. Following the congestion, dilatation and proliferation of the superficial capillaries become apparent. Accompanying the blood vessel engorgement are varying degrees of pustulation and subcutaneous and subcuticular hypertrophy. Seelig's comparison of its likeness to a sponge is a particularly good one. The process may continue until the actual cartilaginous structures of the nose may be deformed. Sanvenero-Rosselli [*120*], Ollier, and Joseph [*70*], all reported cartilages invaded and destroyed by rhinophyma; whereas Rees reports the cartilages were simply pushed aside and were normal.

Rhinophyma usually affects the lower two thirds of the nose. The nose, ears, forehead and chin may be involved. It must be emphasized that the transformation from acne rosacea to rhinophyma is one of many years—an extremely slow one. (Fig. 8.)

Numerous interesting theories have been advanced as the cause of rhinophyma. Demodex folliculorum has been found in numerous cases studied by one author. He reported an improvement in this disease through the use of antiparasitic agents. His conclusions have never

been confirmed or denied by any other worker in this field [3].

Cheadle [17] presents a long discussion on acne rosacea in which he states (as many in that day believed) that acne rosacea is definitely associated with excessive alcohol, gastric and uterine disorders and prolonged exposure to heat and cold. Although this article does not mention rhinophyma by name, there are several statements referring to fibrous growth associated with acne rosacea. He further states: "The essential fact that is associated in hyperplasia appears to be *prolonged* engorgement of the vessels of the part and increased pressure in the capillaries. Sir W. Kenner first drew attention to the fact that this engorgement increases the interstitial exudation of the lymph which becomes organized into fibrous tissue. The lymph so effused is either converted into fibrous tissue or the connective tissue already existing is stimulated by this excessive supply of pablum to excessive growth."

Lassar suggests this disease is a congenitally predisposing one, and appears in the so-called "wide pored" individual. The substitution of cleansing creams for soap and water and also focal infections have been suggested by Dinsmore et al. [25] as contributing to the development of this disease. Von Bruns [146] states that acne rosacea and rhinophyma have nothing in common. He attributes the nose hypertrophy to growth of congenital anlage. Angioneurosis was explained by Kaposi as the cause of growth of connective tissue, of dilatation of blood vessels and of degeneration of sebaceous glands.

Tulipan states that most cases of acne rosacea are due to a deficiency of vitamin B complex. Vitamin C deficiency has also been listed as a possible cause. Being a "seborrheic type of individual" was listed by Engman as the cause of this disease; thus, he considered rhinophyma as a familial disease representing some type of hereditary transmission. Mangabeira-Albernaz [89] is of the opinion that the disease may be caused by chronic sores with accompanying local vascular injury. Ohmann-Dumesnil, Hans Hebra, Franz Wings, and Wende and Wende [152], among many, have elaborate discussions of pathology and descriptions of the microscopy of rhinophyma. Historically, the disease was first designated as rhinophyma by Hebra in 1845. Pennoyer observed that most of these patients come from the "poor class." We consider the "poor class" due to rhinophyma—not rhinophyma due to the "poor class."

Wende and Wende in describing a patient write, "His habits, excepting his unquenchable thirst, are good, his fondness for beer is especially well established. It is his custom, during the hours of employment, rather to palliate the morbid desire for drink than to be confused by the uncertainty of taking too much. Productiveness becomes the result of submission. Disposed to joy and pleasure, cares disappear, and, dissatisfied with the drugging of the week, he consecrates Saturday evening and Sunday, by taking a full course, to enjoy relaxation suitable to his means and peculiarities. His bibulous propensities were contracted early in life, and have continued to the present time, yet, in defiance to this manner of living, he was always industrious and never indolent. The unwisdom of these beery indulgences at fixed periods often induced intestinal distress and diarrhea, which were additional penalties, in his case, for violation of nature's law. Nature is relentless, and knows nothing of extenuating circumstances; she never forgets and she never forgives."

The exact cause other than it is the end product of acne rosacea is not known. The proportion of acne rosacea occurring in men and women is 1:3; whereas, the proportion of the occurrence of rhinophyma in men and women is reported to be 12:1. The exact reason for this paradox has never been answered. Numerous predisposing factors have been blamed—heredity, chronic alcoholism, gastronomic excesses, immoderate use of coffee and tea, chronic stomach trouble, intestinal derangement, certain forms of heart trouble and repeated external irritations from cold and heat have all been mentioned but no agreement is noted. Most authors agree, however, that alcohol has nothing to do with the cause and development of rhinophyma. Numerous cases have been reported in total abstainers.

Wolfe [154] writes that, "Philadelphia, which is known to have a high percentage of alcoholics, turned up but one case of rhinophyma." He suggests that the preference of the disease for men in their fifties may be due to an excess of androgenic substances. Wolfe bases his opinion on experimental research carried out by Hamilton which shows that introduction of androgenic hormones generates the growth of sebaceous glands in the development of acne rosacea.

There have been no important advances in our knowledge in finding the cause of this disease since the original theories advanced in 1850 by German authors.

"THE SHAVERS" VS. "THE GRAFTERS"

Of the many diseases treated in medicine none can be as rewarding to the physician as the satisfactory treatment of rhinophyma. There are many methods of treatment advocated. Basically these are "the shavers" and "the grafters."

Electroshaving, chemicals, dermabrasion, excision and replacement with a full thickness graft, split thickness grafts, injection of fibrolysin, mercury vapor, ultraviolet rays, x-rays, liquid nitrogen, liquid oxygen, radium, carbon dioxide snow and even ACTH and cortisone have all been advocated during the past century.

Although roentgenization may give good results in acne rosacea by destroying or inhibiting the sebaceous and oily characteristics of the skin, it is generally agreed it is totally inadequate for a true case of rhinophyma. In spite of this, there is an occasional appeal for this treatment heard today. Those who advocate x-ray therapy, of course, are in the small minority and display no photographic evidence to support their advocation of x-ray treatment.

Conflicting statements are seen in the literature primarily due to satisfactory results obtained by most methods. Therefore, all other methods are rejected by the authors. A textbook on this subject as recent as 1956, and repeated in a recent article, contains the following: "A thorough study of domestic and foreign literature discloses many medical surgical methods of reconstruction of rhinophyma ranging from superstitious rituals to partial excision of the nose. For many years we operated on patients for their rhinophymatous deformity by employing two surgical methods described in the literature without acceptable, functional or cosmetic results. Some authors advocated excision of the rhinophymatous tissue leaving the area denuded and expecting the denuded surface to epithelialize and produce a satisfactory skin covering. In our opinion, based on experience, this conception of skin regeneration is a fallacy."

We believe, of course, that the preceding is *not a fact*. This same author does not even mention planing or electrocautery for the treatment of rhinophyma in the portion of the book he contributed. He has summed up the objections to "the shavers" as follows: "The nose occupies the most prominent location of the face, is subject to trauma, cold weather and burning of the skin. Correction by means of epithelialization and fibrous tissue by split skin graft will produce a nose abnormal in color, etc." We challenge these and other advocates of grafting to try any of the other methods and discover that equally good results will be obtained—in fact, better—cosmetically.

FIG. 9. A, rhinophyma invading the entire nose; B, the disaster of overtreatment [*87*].

Of all the methods of treatment, only one case of a recurrence has been reported regardless of the modality used in the cure of rhinophyma. Edelstein's case [*34*] was a recurrence following a dermabrasion technic.

Some authors still advocate split and sliding grafts for the correction of rhinophyma. Lewis [*80*] has answered them well: "It is my opinion; however, that in rhinophyma the integument

A B

FIG. 10. A, preoperative front view; B, lateral view, July 22, 1958.

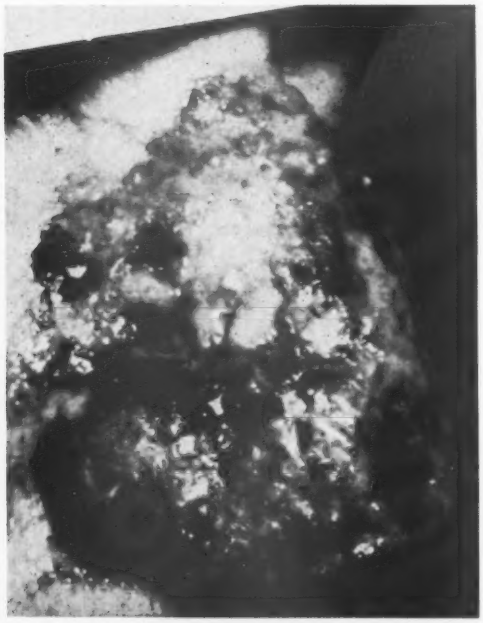


FIG. 11. Immediate postoperative view, July 22, 1958.

of the nose and that surrounding the lesion is considerably degenerated—too much so to furnish adequate material for (proper) skin flaps."

The essence of treatment is simply the excision of the hypertrophic sebaceous glands and connective tissue by shaving or whittling to a nose of normal shape; the method of this excision is not important. If the cartilages are exposed, irregularities or snarling expressions will be produced. When this happens, of course, skin grafting has to be done. Only one such case has been reported in the entire literature.

It is our opinion based on the actual photographic results printed in the literature that cauterization is probably the method of choice in accomplishing this sculpturing as bleeding is easier to control. As in any method, care must be taken not to injure the cartilages.

Von Bruns has pointed out that grafting often leads to the development of retention cysts underneath the graft, with a subsequent breaking through and ulceration.

Smith [*132*] has recently given the historical evolution of "the grafters." We do not agree

A B

FIG. 12. A, three days postoperatively; B, three weeks postoperatively.

with his conclusions, but for students of this subject the many details and various technics of "the grafters" are given. His article implies that many catastrophic results from electroshaving have occurred; actually, only one is reported. He vehemently condemns "the shavers" without ample clarification; stating that, "Such treatment may end disastrously and entail additional plastic operations for reconstruction as well as the added expense and misery to the patient." This may be true; however, if one errs with the shaving technic, this reconstruction is as simple as it would have been using "the grafters'" technic first. Nothing is lost. We were happy to see this same author agree that "the shavers" were the most widely followed in modern day surgery, which is another argument, we think, in favor of "the shavers."

The actual technic is simple. It is important not to injure the cartilage. One must leave enough soft tissue to allow re-epithelialization of the nose. This occurs readily and smoothly. The most difficult portion of the surgery for the novice is finding the exact depth to shave the nose. Also, what did the person's nose actually look like before surgery? Having a prerhinophymatous photograph of the patient is of considerable help. One of our most difficult patients was a person whose nose had a marked deviation from the norm which was hidden by the tremendous hypertrophy and was not actually noticed until healing had occurred. It made no difference whatsoever in the final results (we had considered doing a rhinoplasty); however, the patient is happy with his present nose.

FIG. 13. Six weeks postoperatively.

Skin grafting has many risks, namely, failure, necrosis, infection and latent ulceration; it is extremely tedious, and most important is not as perfect cosmetically. This last point is especially proved if one takes time out to review the end results reported in the literature. There is, also, more discomfort in grafting as there is pain in a donor site as pointed out by Lewis [*80*].

The most important fact is that the shaving method is so much easier, both for the surgeon and also for the patient. Why bother with all the fuss of grafts, etc., especially when the arguments of "the grafters" is not borne out either by actual experience or fact. They contend that the epithelialization is not tough enough for the elements or that there is no

FIG. 14. Six months postoperatively. A, lateral view; B, front view.

such thing as epithelialization, etc., etc. We dare them to try one case with proper supervision and they will stop all this tedious work of forming grafts, making molds for proper pressure, etc. and obtaining, in general, poorer results. "The grafters" all seem to be a little shy about showing good profile and full face photographs (there are some exceptions). It has been our impression that "the shavers" have not been as guilty of hiding their results.

Regardless of the method chosen, with the exception of partial shaving methods, the use of surgery for this condition is sanguineous in its results. This is particularly true of the "cold" technics. We have found, even with cautery methods, there is considerable bleeding. This can be controlled more readily by placing one finger in the nostril and applying counterpressure. We have not had much experience with topical adrenalin and other hemostatics. In general, the bleeding is of a very short duration.

For most patients local anesthesia is all that is required. Most surgeons prefer to perform this operation as an office procedure. On the other hand, some surgeons require several days' hospitalization and employ general anesthesia. We think this is entirely up to the operator.

In summary, most surgeons use the shaving method of treatment. Each surgeon is very vehement as to his particular method being superior. "Whittling" gives better results cosmetically whether done "cold" or "hot" when viewed historically. Grafting has perhaps only one advantage, namely, it may be more resistant to the elements.

COMPLICATION OF THERAPY

Although the treatment of rhinophyma is basically simple, the degree of skill required is clearly shown in one complication reported in the literature. This is Maliniak's famous reconstructive case [*87,88*]. This case was referred to him for reconstruction after excessive treatment. Of course, this is a terrible disaster and it is the reason many "shavers" proceed slowly and give multiple treatments. We believe this could easily be avoided by using the finger intranasally. This would also provide an index to the degree of heat being produced. Maliniak reconstructed this case with a tube graft with excellent results.

WOMEN AND RHINOPHYMA

Bechet [6] is given credit for the first reported case of rhinophyma in women. Numerous articles prior to 1926 discuss the incidence of this disease in women, but this is the first which meets our requirement of proved rhinophyma. Ginsburg [52]; Rosenburg and Felsher [115]; Sams (twenty-one year old woman); Eller [36], Lewis [80] and Galtier [48] have added six additional cases making a total of eight proved cases of this disease in women.

Most textbooks and articles on this subject report a ratio of men to women as 12:1. Using only proved cases in accordance with our original requirements, we have reviewed this ratio and find it to be 20:1. We think this is probably a more accurate figure as it is based on the largest possible number of reported cases.

MALIGNANT DEGENERATION AND RHINOPHYMA

Dr. Novy's case [102] of squamous cell carcinoma with an associated basal cell carcinoma on the cheek was the first case occurring in rhinophymatous tissue to be reported. Eisenklam [35], Novy and Rees (two cases) and Professor Newman of Vienna have all reported this phenomenon bringing to a total five cases thus far reported. All but one of Rees's cases were squamous cell; one was a basal cell carcinoma.

Two cases of probable rhinophyma are described that are related to lymphomas. One by Ayres and Jensen [4] is questionable, both in regard to being a true rhinophyma and also as to the lymphoma, since the patient was cured of both conditions by hormone therapy. To our knowledge this cure has never been duplicated for either disease. The second case is described by Calvert and Smith [15] and appears to be a true case of both rhinophyma and lymphoma. In regard to Ayres' article, it is suggested that rhinophyma may be related to a deficiency of male hormones. His reasoning is based on the fact that acne rosacea has been related by others to hormonal changes in menopause. Also, rhinophyma occurs predominantly in men past middle age.

Rees suggests if more pathologic studies were conducted, more malignant degeneration would be seen.

CASE REPORT

E. L., sixty-two years of age, had progressive enlargement of his nose and a "rash" of both cheeks for five years. History and results of physical examination were characteristic of rhinophyma. Except for severe pyorrhea, the physical examination revealed data essentially within normal limits.

On July 22, 1958 the patient was subjected to electroshaving procedures for the correction of his rhinophyma. This was accomplished under local Lidocaine blockage of the nose and cheek regions. This procedure was carried out in the office in less than thirty minutes. Codeine (gr. ½) was the only analgesic required during the immediate postoperative period. The accompanying photographs (Figs. 9–14) can more accurately describe the results. The patient was seen eighteen months later and was free of disease and feeling well.

SUMMARY

1. A plea is made to encourage patients with rhinophyma to get proper treatment.

2. The history, etiology, malignant degeneration and management of rhinophyma is discussed.

3. The ratio of 20:1 of men to women is probably more accurate than the usually stated 12:1 ratio.

4. A case report is presented.

REFERENCES

1. Andouin, P. Rhinophyma in medicine and classical art. *Rev. laryng.*, 80: 461, 1959.
2. Aschoff, I. Tratado de Anatomia Patologica, vol. II. Madrid, 1934. Labor S. A.
3. Ayres, S., Jr. and Anderson, N. P. Acne rosacea. I. Response to local treatment for Demodex folliculorum. *J. A. M. A.*, 100: 645, 1933.
4. Ayres, S., Jr. and Jensen, A. K. A case for diagnosis (lymphoblastoma?). *Arch. Dermat. & Syph.*, 49: 457, 1944.
5. Ayres, S., Jr. and Jensen, A. K. Otorhinophyma, pruritis and alopecia totalis syndrome. *Arch. Dermat. & Syph.*, 56: 379, 1947.
6. Bechet, J. Rhinophyma in a woman. *Arch. Dermat. & Syph.*, 14: 86, 1926.
7. Behcget, H. Behandlung des Rhinophyma. *Dermat. Wchnschr.*, 88: 129, 1929.
8. Berson, M. I. Rhinophyma. *Plast. & Reconstruct. Surg.*, 3: 74c, 1948.
9. Blasi, R. Roentgenotherapy in rhinophyma. *Gior. ital. di mal. esot. e. trop.*, 7: 16, 1934.
10. Bordier, H. Effective use of electric coagulation in rhinophyma. *Presse méd.*, 38: 535, 1930.
11. Bourgoin, E. Plastic Surgery. London, 1933. Medical Publications Ltd.
12. Buorquet, J. La Veritable Chirurgie Esthetique du Visage. Paris, 1936. Librairie Plon.
13. Burgess, L. H. Acne punctata and acne rosacea or copper nose. *London M. Gaz.*, 9: 314, 1849.
14. Burks, J. W. Wire Brush Surgery. Springfield, 1956. Charles C Thomas.
15. Calvert, R. J. and Smith, E. Metastatic acrophacy in lymphatic leukemia. *Blood*, 10: 545, 1955.

16. Campos, R. Combined use of surgery and radium in the treatment of rhinophyma. *Ars méd., Barcelona*, 12: 85, 1936.
17. Cheadle, W. R. Acne rosacea. *Practitioner*, 73: B-1, 1874.
18. Cinelli, J. A. Rhinophyma. *New York J. Med.*, 40: 1672, 1940.
19. Clark, W. L. The treatment of rhinophyma by the dessication method. *Urol. & Cutan. Rev.*, 25: 63, 1921.
20. Coldrey, R. S. Treatment of rhinophyma. *Brit. J. M.*, 2: 518, 1930.
21. Crabtree, W. C. Rhinophyma. *California & West. Med.*, 45: 485, 1936.
22. Darier, J., Civatte, A. and Tzanck, A. Prescis de Dermatologie, 3rd. ed. Paris, 1947. Masson & Cie.
23. Ducourtioux, M. Rhinophyma and its treatment. *Presse mêd.*, 47: 799, 1939.
24. Desouttes, L. Technique of surgical therapy of rhinophyma. *Tech. chir.*, 25: 177, 1933.
25. Dinsmore, R. S. and Keller, K. W. Rhinophyma. Report of a case. *Cleveland Clin. Quart.*, 5: 217, 1938.
26. Dohi, K. Ein Beitrag zur Kenntriss des Rhinophyma. *Arch. f. Dermat. u. Syph.*, vol. 37, 1896.
27. Dorigo, L. Use of electric bistoury for removing rhinophyma. *Bollettino*, 11: 62, 1937.
28. Dubreuilk, W. L'acne hypertrophique due nez et son, traitment chirurgical. *Ann. dermat. et syph.*, 4: 785, 1903.
29. Dufourmental, L. Chirurgie Reparatrice et Correctrice des Teguments et des Formes. Paris, 1939. Masson & Cie.
30. Duhring, L. A. Rosacea, acne rosacea, gutta rosea, rhinophyma. *Photographic Rev. Med. & Surg.*, 2: 32, 1871.
31. Dyer, I. A case of hypertrophic rosacea (pachydermatosis) resembling tubercular leprosy, cured with thyroid extract. *New Orleans M. & S. J.*, 49: 402, 1896.
32. Dyer, I. A case of hypertrophic rosacea (pachydermatosis) resembling tubercular leprosy, cured with thyroid extract. *Tr. Am. Dermat. A.*, pp. 33–35, 1896.
33. Dyer, I. Preliminary report on a case of hypertrophic rosacea resembling tubercular leprosy. *Tr. Louisiana M. Soc.*, pp. 295–300, 1896.
34. Edelstein, J. Recurrent reaction following dermatologic abrasion. *Arch. Dermat.*, 1954.
35. Eisenklam, D. Rhinophyma; case of carcinomatous degeneration with remarks on the treatment. *Wien. klin. Wchnschr.*, 44: 1407, 1931.
36. Eller, J. J. A simple procedure for the cure of rhinophyma. *New York J. Med.*, 33: 741, 1933.
37. Ellis, A. Specimen from a case of rhinophyma. *Proc. Path. Soc., Philadelphia*, 7: 238, 1903.
38. Epstein, E. Skin Surgery. Philadelphia, 1956. Lea & Febiger.
39. Farina, R. Rhinophyma. Plastic correction. *Plast. & Reconstruct. Surg.*, 6: 461, 1950.
40. Federspiel, M. N. Rhinophyma with report of case. *Internat. J. Orthodontia*, 18: 92, 1932.
41. Federspiel, M. R. Rhinophyma with report of case. *Wisconsin M. J.*, 29: 75, 1930.
42. Fischkin, E. A. The treatment of rhinophyma. *Am. J. Clin. Med.*, vol. 20, 1913.
43. Fiumcelli, F. Rhinophyma: diathermocoagulation case. *Dermosifilografo*, 9: 35, 1934.
44. Fiumcelli, F. Rhinophyma: diathermocoagulation case. *Minerva med.*, 1: 228, 1934.
45. Foss, M. H. Surgical treatment of rhinophyma with diathermic snare. *Finska norske laegeforen.*, 78: 1123, 1958.
46. Fowler, R. S. Rhinophyma. Operative treatment. *Am. J. Surg.*, 25: 258, 1911.
47. Fuld, J. E. The surgical treatment of rhinophyma. *J. A. M. A.*, 72: 1734, 1919.
48. Galtier. Le traitement du rhinophyma. *M. Acad. chir.*, 85: 436, 1959.
49. Gaskoin, F. Clinical notes on acne rosacea. *M. Press & Circ.*, 15: 352, 1873.
50. Gibbon, J. H. Rhinophyma. *Ann. Surg.*, 46: 320, 1907.
51. Gillies, H. and Millard, R., Jr. The Principles and Art of Plastic Surgery. Boston, 1957. Little, Brown & Co.
52. Ginsburg, L. Rhinophyma in a woman. *Arch. Derm. & Syph.*, 32: 468, 1935.
53. Glott, M. A. Rhinophyma. *Illinois M. J.*, 61: 1745, 1932.
54. Gottheil, W. S. Rhinophyma. *Tr. Pan-Am. M. Congr.* 1893, 2: 1683, 1895.
55. Grattan, J. F. Rhinophyma. *J. A. M. A.*, 74: 1450, 1920.
56. Grattan, J. F. Rhinophyma. *M. Rec.*, 98: 665, 1920.
57. Grattan, J. F. Rhinophyma. A report of six cases cured by radical operation followed by x-ray and acid treatment to relieve the associated hypertrophy of the skin. *Surg. Gynec. & Obst.*, 41: 99, 1925.
58. Gurdin, M. and Pangman, W. J. A simple electrosurgical treatment of rhinophyma. *California Med.*, 73: 171, 1950.
59. Hanradan, E. M., Jr. The surgical treatment of rhinophyma and report of a case. *Bull. John Hopkins Hosp.*, 32: 49, 1921.
60. Haret, M. Rhinophyma: Case cured by radiotherapy. *Bull. et mêm. Soc. de radiol. méd. de France*, 16: 236, 1928.
61. Heineck, A. P. A case of rhinophyma successfully treated by decortication. *J. A. M. A.*, 62: 105, 1914.
62. Hollaender, E. Die Karikatur und Satire in der Medizin. Stuttgart, 1905. Ferdinand Enke.
63. Hunt and Chambers Papers. *Lancet*, 1846; also condensed in the 14th number of Braithwaite's retrospect.
64. Hunt, H. A. A case of rhinophyma. *Laryngoscope*, 43: 282, 1932.
65. Hebra, H. Quoted by Jewett [*68*].
66. Israel, S. Rhinophyma. Report of a case. *Laryngoscope*, 32: 218, 1922.
67. Jamieson, W. A. Case of hypertrophic acne rosacea. *Tr. Med.-Chir. Soc. Edinburgh*, 23: 225, 1903.
68. Jewett, P. A. Acne rosacea: third degree. Great thickening of the skin of the nose with development of fleshy excrescences, treated by excision. *M. Rec.*, 15: 371, 1879.

69. JORGE, J. M. Results of nasal decortication performed 20 years ago. *Bol. y trab., Soc. de cir. de Buenos Aires*, 20: 472, 1936.
70. JOSEPH, J. Nasenplastik und sonstige Geischtsplastik nebst Mammaplastik. Leipzig, 1931. Curt Kabitzsche.
71. KEEN, W. W. Rhinophyma: report of a case with operation for its relief. *Ann. Surg.*, 39: 665, 1904.
72. KEEN, W. W. Rhinophyma: report of a case with operation for its relief. *Tr. Phila. Acad. Surg.*, pp. 16–19, 1905.
73. KELLOG, A. O. Case of acne rosacea treated with liquor potassac arsenstis. *Buffalo M. J.*, 3: 598, 1847.
74. KIRSCHNER, M. Tratado de Taenica Operatoria General y Especial, vol. II. Madrid, 1940. Labor S. A.
75. KLAUDER, J. V. Rhinophyma. Treatment by electrodesiccation. *Arch. Dermat. & Syph.*, 33: 885, 1936.
76. KULVIN, M. M. Surgical treatment of rhinophyma. *Eye, Ear, Nose & Throat Month.*, 35: 715, 1956.
77. KYRLE, J. Rhinophyma: a histological study. Also a contribution to the question of post-fetal sebaceous gland and hair formation. *Urol. & Cutan. Rev.*, 23: 311, 1919.
78. KYRLE, J. Rhinophyma. *Urol. & Cutan. Rev., Tech. Supp.* 1(3): 276, 1913.
79. LEDO, E. Rhinophyma. Technique of plastic surgery. *Acta dermo-sif.*, 27: 249, 1934.
80. LEWIS, G. K. Rhinophyma. *Plas. & Reconstruct. Surg.*, 24: 190, 1959.
81. LIEBERTHAL, D. Treatment of rhinophyma. *Am. J. Clin. Med.*, 20: 686, 1919.
82. LOWENTHAL, G. Rhinophyma: surgical excision following oblique cutaneous incision. *Tr. Am. Acad. Opth.*, 57: 625, 1953.
83. MACKEE, G. M. X-rays and Radium in the Treatment of Diseases of the Skin, p. 422. Philadelphia, 1938. Lea & Febiger.
84. MACNAUGHTON, B. F. Report of a case of rhinophyma. *Canad. M. A. J.*, 24: 271, 1931.
85. MACOMBER, D. W. Surgical cure of acne rosacea and rhinophyma. *Rocky Mountain M. J.*, 43: 466, 1946.
86. MALBEC, E. F. Rhinophyma: results of surgical therapy. *Cong. latino am. cir. plast.*, 4: 355, 1949.
87. MALINIAK, J. W. Rhinophyma: treatment and its complications. Soc. Scient. Francaise de Chir. Repar. Plast. et Esthet. *Compt. rend. Acad. d. sc.*, pp. 250–257, 1930.
88. MALINIAK, J. W. Rhinophyma. Its treatment and complications. *Arch. Otolaryng.*, 13: 270, 1931.
89. MANGABEIRA-ALBERNAZ, P. Rhinophyma. Pathogenesis and therapy. *Rev. Laryng.*, 56: 1199, 1935.
90. MARIN, A. Rhinophyma and its treatment. *Canad. M. A. J.*, 58: 69, 1948.
91. MARIN, A. A few remarks about the treatment of rhinophyma. *Canad. M. A. J.*, 25: 589, 1931.
92. MARTIN, A. Rhinophyma therapy by Ollier's surgical decortication. *Bull. Soc. franç. dermat. et. syph.*, 46: 705, 1939.
93. MASON, M. L. and ALLEN, H. S. Surgical correction of rhinophyma. Case report. *Quart. Bull. Northwestern Univ. M. School*, 25: 60, 1951.
94. MILLIGAN, W. The histological features of rhinophyma. *Proc. Roy. Soc. Med.*, 9: 77, 1916.
95. MILLIGAN, W. Rhinophyma: its etiology, pathology and treatment. *Lancet*, 2: 643, 1915.
96. MORESTIN, H, LEDENTU, A., DELBERT, P. and CHEUASSU, M. Affections Chirurgicales de la Face. Paris, 1911. Bailliere.
97. MORRIS, J. On the treatment of acne rosacea. *Lancet*, 1: 236, 1855.
98. NETO, J. R. Rhinophyma with special reference to surgical therapy. *Rev. oto-laring. de São Paulo*, 4: 8, 1936.
99. NEW, G. B. Rhinophyma. *Clin. North. America*, 1: 1393, 1921.
100. NEW, G. B. Rhinophyma. *Laryngoscope*, 29: 391, 1919.
101. NIEDELMAN, M. L. Rhinophyma. Treatment by electro-shaving. *Arch. Dermat. & Syph.*, 70: 91, 1954.
102. NOVY, F. G., JR. Rhinophyma with carcinomatous degeneration. *Arch. Dermat. & Syph.*, 22: 270, 1930.
103. NYLEN, B. Operative treatment of rhinophyma. *Acta Soc. med. upsalien.*, 62: 17, 1957.
104. OHMANN-DUMESNIL, A. H. A case of rhinophyma: operation. *St. Louis M. & S. J.*, 59: 265, 1890.
105. OHMANN-DUMESNIL, A. H. Rhinophyma. A case in which operation produced a good result. A new pathological condition found. *Internat. M. Mag.*, 3: 24, 1894.
106. OHMANN-DUMESNIL, A. H. A case of rhinophyma: operation. *St. Louis Clin. Phys. & Surg.*, 3: 575, 1889.
107. OHMANN-DUMESNIL, A. H. A case of rhinophyma. *Quart. Atlas Dermat.*, 3: 87, 1895.
108. OHMANN-DUMESNIL, A. H. A case of rhinophyma. *Tr. Pan-Am. M. Cong.*, 2: 1688, 1893.
109. PENNOYER, G. P. Rhinophyma. *S. Clin. Nortb America*, 16: 885, 1936.
110. PERL, M. Diathermy treatment of rhinophyma. *Prat. du Nord de l'Afrique*, 5: 150, 1932.
111. PIFFORD, H. G. Acne and acne rosacea. *M. Rec.*, 14: 161, 1878.
112. POSSE, R. P. Cirurgia Estetica. Buenos Aires, 1946. Miguel Violetto.
113. REES, R. D. Basal cell carcinoma in association with rhinophyma. *Plast. & Reconstruct. Surg.*, 16: 282, 1955.
114. RIORDAN, T. J., CANIZARES, O. and MORRIS, G. E. Folliculitis of the chin with enlargement resembling rhinophyma. "Mentophyma." *Arch. Dermat. & Syph.*, 45: 963, 1942.
115. ROSENBERG, W. A. and FELSHER, I. M. Rhinophyma and acne rosacea treated with the electrosection current. *Illinois M. J.*, 97: 281, 1950.
116. ROHRBACH, R. Compendio de Dermatoligia, Rio de Janiero, 1943. Cientifica.
117. SAFIAN, J. Corrective Rhinoplastic Surgery. New York, 1935. Paul B. Hoeber.
118. SALINGER, S. Rhinophyma: operation, treatment of gigantic case. *Arch. Otolaryng.*, 11: 620, 1930.
119. SAMS, W. M. Rhinophyma, with unusual involvement of chin. *Arch. Dermat. & Syph.*, 26: 834, 1932.
120. SANVENERO-ROSSELLI, G. Chirurgia Plastica del Naso, vol. IX, p. 2. Rome, 1931. Luigi Possi.

121. Scott, M. J. Rhinophyma: a procedure for office treatment. *Northwest Med.*, 55: 46, 1956.
122. Seelig, M. G. Rhinophyma. *Surg. Gynec. & Obst.*, 30: 394, 1920.
123. Seelig, M. G. Rhinophyma. *S. Clin. North America*, 5: 1381, 1925.
124. Seelig, M. G. Rhinophyma. *Tr. West. S. A.*, 29: 241, 1919.
125. Seltzer, A. P. On a hereditary factor in rhinophyma with associated acromegalic condition. *M. World*, 61: 310, 1943.
126. Seltzer, A. P. The effect of ACTH in the surgical treatment of acne and rhinophyma. *Am. Pract. & Digest Treat.*, 2: 882, 1951.
127. Sheehan, J. E. A case of rhinophyma and its cure. *M. J. & Record*, 119: 94, 1924.
128. Sheehan, J. E. and Swanker, W. A. Rhinophyma. *New York J. Med.*, 50: 2841, 1950.
129. Shoemaker, J. V. Rosacea. *Tr. M. Soc. Pennsylvania*, 12: 116, 1878.
130. Simard, L. C. Note on the pathology of rhinophyma. *Canad. M. A. J.*, 58: 70, 1948.
131. Shanturov, A. G. Plastic surgery of the nose with a split skin flat after removal of rhinophyma. *Vestnik oto-rino-laringologii*, 21: 88, 1959.
132. Smith, A. E. Correction of advanced rhinophyma by means of plastic reconstructive surgery. *Am. J. Surg.*, 96: 792, 1958.
133. Spillman, L., Hamant, A. and Watrin, J. Surgical therapy of rhinophyma. *Bull. Soc. franç. dermat. et sypb.*, 41: 813, 1934.
134. Squire, B. A case of rhinophyma after operation. *Tr. Clin. Soc. London*, 24: 238, 1901.
135. Squire, B. On Rhinophyma or "The Hammer Nose." London, 1889. J. & A. Churchill.
136. Squire, B. Acne rosacea treated by ointment of chrysophanic acid. *M. Times & Gaz.*, 1: 665, 1877.
137. Startin, J. Treatment of acne rosacea. *Brit. M. J.*, 1: 932, 1878.
138. Startin, J. Treatment of acne rosacea. *Lancet*, 1: 925, 1875.
139. Stein, D. J. Case of rhinophyma. *Ann. Otol. Rhin. & Laryng.*, 25: 508, 1916.
140. Stelwagon, H. W. A Treatise on Diseases of the Skin, 9th ed. Philadelphia, 1921. W. B. Saunders Co.
141. Stokes, J. and Beerman, H. Effect of skin on emotional and nervous states. IV. The rosacea complex. *Arch. Dermat. & Syph.*, 26: 457, 1932.
142. Strong, B. F. Rhinophyma, an exaggerated case. *Am. J. Clin. Med.*, 20: 685, 1913.
143. Tamerin, J. and Bornstein, L. A. Trichloracetic acid in the management of rhinophyma. 3: 28, 1950.
144. Thorek, M. Modern Surgical Technic. Philadelphia, 1949. J. B. Lippincott Co.
145. Villa-fuerte, C. F. Plastic surgery for rhinophyma. *J. Philippine M. A.*, 31: 68, 1955.
146. von Bruns, P. Ueber das Rhinophyma. *Beitr. z. klin. Chir.*, vol. 391, 1903.
147. Wagner, C. Case of acne rosacea or hypertrophic outgrowths removed by operation. *Arch. Clin. Surg.*, p. 21, 1876.
148. Watten, J. C. and White, C. J. The treatment of rhinophyma. *St. Paul M. J.*, 5: 887, 1903.
149. Wehr, R. H. Rhinophyma. Case report. *Arch. Otolaryng.*, 50: 433, 1949.
150. Wende, G. N. and Bentz, C. A. Rhinophyma. *Tr. Am. Dermat. A.*, 28: 15, 1904.
151. Wende, G. W. and Bentz, C. A. Rhinophyma: a pathological analysis and five separate tumors occurring in the same patient. *Cutan. Dis.*, 22: 447, 1904.
152. Wende, E. and Wende, G. W. Rhinophyma. *Buffalo M. J.*, 39: 207, 1899.
153. Whitham, J. D. Unusual case of rhinophyma. *New York J. Med.*, 38: 1233, 1938.
154. Wolfe, M. M. Rhinophyma with new etiologic and therapeutic considerations. *Laryngoscope*, 53: 172, 1953.
155. Wood, J. C. Rhinophyma. *Surg. Gynec. & Obst.*, 15: 622, 1912.

Untimely Lymph Node Biopsy

HAYES MARTIN, M.D., *New York, New York*

THE PURPOSE of this communication is to call the attention of the medical profession in general not only to the needlessness but also to the possible harmfulness of *excisional* lymph node biopsy as *the first* or even as an early step in the diagnosis of cancer. It is hereby proposed that the foregoing procedure be withheld and postponed until all other diagnostic measures have failed. In brief, excisional or incisional lymph node biopsy should be used only as a last resort and then preferably by the surgeon who accepts the responsibility for treatment himself if the diagnosis eventually proves to be cancer. It is not intended that the foregoing admonition should apply to aspiration biopsy [1], a safe and harmless measure which does not unfavorably alter the clinical setting for subsequent treatment and which, when specifically indicated and in the hands of an experienced clinician and pathologist, has many applications in problematic cases.

It has long been considered requisite to a thorough general physical examination to note and record the presence of any palpable superficial lymph nodes, particularly in the cervical, axillary, epitrochlear and inguinal regions. Even slight palpability is considered by some physicians as being suspicious, while definite enlargement is rather generally regarded as positive evidence of involvement by disease, either neoplastic or inflammatory.

In this connection, doctors of medicine, who were graduated thirty to forty years ago and served internships in teaching hospitals, can probably recall that it was then rather generally considered a commendable sign of diagnostic acumen in a problematic case to excise promptly (or to refer the patient for such an excision) of such enlarged lymph nodes (commonly referred to as "glands") for histologic examination. It is particularly relevant to the present discussion that, during this period, once the node had been excised and the stitches removed, all positive action and practical interest ceased in the majority of cases, actually not by design or oversight but rather as the inevitable consequence of several unrelated human factors. To begin with, it could be a week or ten days before a routine histologic report was returned and specifically noted on the clinical record. By that time, the patient had usually been discharged, and interest in the case had tended to cool, or possibly a diagnosis had already been established by other means. Secondly, since the "lump," often the chief complaint, had been eliminated, it would be natural that the patient conclude that the minor surgical procedure was curative in purpose and apparently had been successful and that no further follow-up or treatment was required. A histologic report of "hyperplasia" or of any specific inflammatory disease was not often of great diagnostic value. On the other hand if, as in most cases, the report was metastatic cancer, the only clinical diagnosis which could be recorded on the hospital chart was, for example, squamous carcinoma or adenocarcinoma of the neck, groin or axilla. These are obviously incomplete and inadequate diagnoses.

In some instances in which a cancerous node had been removed from the neck, any remaining doubt as to the need for further investigation was resolved by classifying the growth as branchiogenic carcinoma. The unsoundness of such a conclusion, except by long term (five years) exclusion of any other alternative (i.e., cryptic primary), has been established clearly by recent clinical investigations [3–5]. In any event, since in those days cancer arising in the body was widely believed to be almost always incurable and therefore ultimately fatal, there seemed to be little reason or benefit for the clinician to stress the importance of further follow-up or treatment. I, who served a period of four years as intern and surgical resident in two large metropolitan hospitals in New York,

clearly remember the aforementioned attitudes on the part of members of the attending staff in general and naturally on the part of those of the house staff.

During the last thirty years, there have been many changes in both the physician's and the layman's attitude toward cancer. Diagnostic and therapeutic methods have been extended and improved so that a diagnosis of cancer no longer carries with it a hopeless prognosis. Nevertheless, the arbitrary and unthinking practice of prompt lymph node biopsy as the first and sometimes the only positive action still persists.

Those who treat cancer of the head and neck must perforce often accept, uncomplainingly, cases of cancer in which an enlarged cervical node has been excised (or incised), thereby rendering more difficult or sometimes even impossible any subsequent clean surgical excision that is, a radical neck dissection [2,5]. The pity is that in most of such patients, the primary lesion is either plainly obvious or at least capable of being found without too much difficulty in the oral cavity, hypopharynx, larynx, nasal cavity, paranasal sinuses, thyroid or even in the skin of the scalp. The height of folly and futility is exemplified by the occasional case of a patient in whom, after a positive biopsy of an obvious cancer of the mouth or pharynx, the referring surgeon has also performed an incisional biopsy of an associated cervical mass in order "to make the diagnosis complete" before referral. What better example could there be of useless surgery?

Cancer is obviously not curable unless the primary lesion can be located and adequately treated. A diagnosis of metastatic cancer anywhere in the body is little more than bad news until the primary lesion is located. Such bad news, unless it ultimately serves to expedite a full diagnosis and proper treatment, does little except lengthen the period during which the patient must live under a sentence of death.

Although on first thought it may seem reasonable that an excisional lymph node biopsy and a diagnosis of metastatic cancer would expedite the discovery of a cryptic primary, the reverse is often the case by reason of the several human factors previously mentioned. Therefore, unless a surgeon feels competent and is willing to treat a primary cancer in the head and neck, he should never excise or incise a cervical node for biopsy. If he feels incapable of treating or would be unwilling to treat the primary cancer if it were later found somewhere in the head and neck (or elsewhere), and would therefore refer the case to another surgeon or to a clinic for cancer if the histologic report of a lymph node biopsy were cancer, then in all conscience and common sense he should refer the case *first and without lymph node biopsy*. If he is tempted to establish the diagnosis of a benign disease by lymph node biopsy, he might well give thought to the fact that experience has shown that *asymmetrical enlargement of one or more cervical lymph nodes in the adult is almost always cancerous and is usually due to metastasis from a cryptic primary lesion most frequently in the upper respiratory or alimentary tracts* and less often below the clavicle.

SUMMARY

An enlarged lymph node should never be excised as the first or even an early step in diagnosis. An extended search for a primary lesion and, if facilities are available, an aspiration biopsy should always precede any consideration of open biopsy. If, as a last resort, a cervical node must eventually be removed for diagnosis, the operation should be performed by a surgeon who is able and willing to treat the primary cancer if it is later found somewhere in the head and neck.

REFERENCES

1. Martin, H. E. and Ellis. E. B. Aspiration biopsy. *Surg. Gynec. & Obst.*, 59: 578, 1934.
2. Martin, H. The treatment of cervical metastatic cancer. *Ann. Surg.*, 114: 972, 1941.
3. Martin, H. and Morfit, H. M. Cervical lymph node metastasis as the first symptom of cancer. *Surg. Gynec. & Obst.*, 78: 133, 1944.
4. Martin, H., Morfit, H. M. and Ehrlich, H. The case for branchiogenic cancer (malignant branchioma). *Ann. Surg.*, 32: 867, 1950.
5. Martin, H. and Romieu, C. The diagnostic significance of a "lump in the neck." *Postgrad. Med.*, 11: 491, 1952.

Pulmonary Embolism

A CLINICAL AND PATHOLOGIC STUDY WITH EMPHASIS ON THE EFFECT OF PROPHYLACTIC THERAPY WITH ANTICOAGULANTS

ROBERT E. HERMANN, M.D., JOHN H. DAVIS, M.D. AND WILLIAM D. HOLDEN, M.D.,
Cleveland, Obio

From the Department of Surgery, Western Reserve University School of Medicine and University Hospitals of Cleveland, Cleveland, Obio.

PULMONARY embolism continues to be a dreaded complication that presents many controversial and unclarified aspects. Although it was initially recognized as a medical complication in the late nineteenth century, the majority of reports in the first decades of this century related it primarily to surgical conditions and the postoperative state [6]. The dramatic and often fatal occurrence of this complication during surgical convalescence has given considerable impetus to studies of its prevention and control. In the past fifteen to twenty years, however, there has again occurred an awareness of its high incidence and association with various medical diseases.

Many aspects of the problem of venous thrombosis and subsequent embolization continue to be controversial. An especially moot point at the present time is the efficacy of anticoagulants, such as the coumarins, and the role venous ligation may play in the prevention of further emboli. G. deTakats has stated, "The surgeon's anticoagulant is heparin . . . we have completely abandoned the use of coumarins [7]." Anlyan and Hart commented, "We have been disappointed with the use of coumarin drugs *and* vein ligations in the prevention of further embolism" [3]. Since the majority of our patients have been treated with anticoagulants, with Dicumarol® as the principal one, this controversy was the stimulus for the study presented herein.

STUDY GROUP

The records of all patients with pulmonary embolism in the University Hospitals of Cleveland during the fifteen year period from 1943 through 1957 were studied. Included were all patients from medical and surgical services for adults (twelve years of age and older), including obstetrics and gynecology; also included were all patients diagnosed clinically and at postmortem examination. A total number of 517 patients was found of which 212 died, a mortality of 41 per cent. In this group, 159 autopsies were performed or 75 per cent.

Incidence. During the fifteen year period, the total number of adults admitted to the University Hospitals was 287,370. Thus, pulmonary embolism was included among the discharge diagnoses in .18 per cent of all admissions. (Table 1.) During this period, 62,506 admissions were in the medical services, while 224,864 admissions were in the surgical services, including general surgery, orthopedics, neurosurgery, ophthalmology, otolaryngology, cardiac surgery, urology, obstetrics and gynecology. The medical services had 305 cases of pulmonary embolism, an incidence of .49 per cent of all admissions. The surgical services had

TABLE 1
INCIDENCE OF PULMONARY EMBOLISM IN UNIVERSITY HOSPITALS FROM 1943 TO 1957

Hospital Admissions		No. of Pulmonary Emboli	Per cent Incidence
Type	No. of Patients		
Medical	62,506	305	.487
Surgical	224,864	212	.094
Total	287,370	517	.180

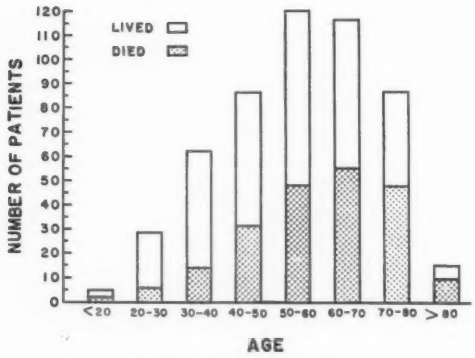


FIG. 1A. Age of patients.

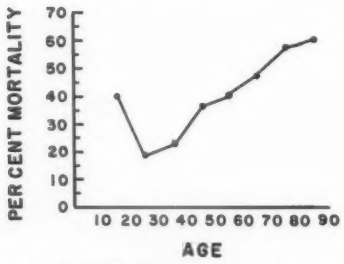


FIG. 1B. Mortality rate per age group.

212 cases, an incidence of .094 per cent of admissions.

Age of Patients. The age range of the patients is depicted in Figure 1A. Mortality per age group is shown in Figures 1A and 1B. It should be noted that the mortality rate increased for each succeeding age group beyond age twenty. The youngest patient in this series was a sixteen year old white boy with severe rheumatic heart disease.

SOURCES OF THE EMBOLI

In the group of 159 patients studied at the time of autopsy, 158 separate thromboses in 127 patients were found which could have been the sources of the emboli. In thirty-two of the patients or 20 per cent, no source for the embolus was found. Twenty-four patients or 15 per cent, had multiple thrombotic sources, any one of which could have been the site of origin of the embolus.

Figure 2 shows the distribution of the sources found. It is noted that thrombi in the heart (two-thirds were in the auricle) were the most demonstrable source. The veins of the leg, thigh, pelvis and the inferior vena cava to-

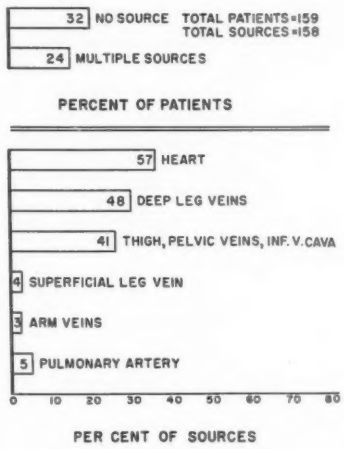


FIG. 2. Source of emboli.

gether accounted for roughly 60 per cent of the sources. The source of the embolus is obviously of great importance when venous ligation is being considered.

Some controversy has arisen recently as to the source of emboli. The earlier literature stated that 60 to 90 per cent of emboli arise from veins of the leg, especially the deep calf veins [*5,13*]. In 1951, McLachlin and Paterson reported a series of dissections of the leg and pelvic veins in 100 autopsies [*11*]. Nineteen of the patients had had pulmonary embolism and thirty-two had evidence of venous thrombosis which was frequently multiple. In this series of dissections, 73 per cent of the thrombi were in the thigh and pelvic veins. More recently, Fowler and Bollinger have reported ninety-seven patients with pulmonary embolism who were examined post mortem; 39 per cent of the emboli arose from the heart, 26 per cent in the pelvis, 20 per cent in the thigh and leg, and in 13 per cent no source was found [*8*]. Our figures tend to support these latter reports.

LOCATION OF FIRST SYMPTOMS

We have found, as have others, that the majority of patients who develop pulmonary embolism have no antecedent signs or symptoms prior to the entrance of the embolus itself which may produce pleuritic pain, cough, hemoptysis, dyspnea, cyanosis, nausea, vomiting, apprehension, fall in blood pressure or sudden death. In this study 356 patients or 70 per cent had these pulmonary symptoms as

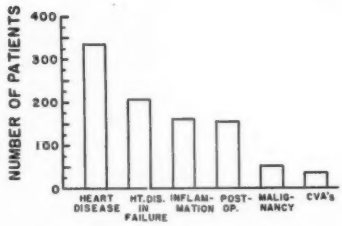


FIG. 3A. Associated conditions.

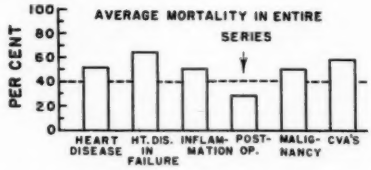


FIG. 3B. Mortality rate with associated conditions.

the initial manifestation of the thromboembolic process. Only 110 patients or 20 per cent had local symptoms or signs of peripheral venous thrombosis prior to embolism. Fifty-one patients had no peripheral or pulmonary symptoms related to thromboembolism. They were in severe heart failure terminating in death; emboli were found at postmortem examination. This is only 10 per cent of the total number of patients, but is one third of all the cases proved at postmortem examination. When clinical diagnosis alone is employed, a significant number of "hidden" pulmonary emboli in patients with an advanced cardiac condition are missed entirely. This emphasizes the great need for more accurate clinical or laboratory examination in these patients. These figures are in accord with those of others who have also reported a preponderance of patients having pulmonary embolism without premonitory signs of venous thrombosis. The results of a survey conducted at the Mayo Clinic indicate this is the case in 75 per cent of their patients [4]. The University of Pennsylvania Hospital reported the same percentage for their patients [15].

ROLE OF ASSOCIATED DISEASE

When the states and conditions of disease associated with pulmonary embolism are studied, much information concerning the incidence, etiology and prognosis can be ascertained. (Fig. 3A.) We have been impressed in this study with the close association of pulmonary embolism with heart disease, especially cardiac decompensation from any cause. Three hundred thirty-five of the patients in this series or 65 per cent had diagnosed heart disease; 201 of these patients were in clinical heart failure. Patients with heart disease accounted for 81 per cent of the deaths in the series. The mortality rate for patients with heart disease in the form of failure was 65 per cent, one and a half times the mortality for all patients with pulmonary embolism. (Fig. 3B.) Most of these patients had the onset of their symptoms during a period of hospitalization for therapy for heart failure. They were, for the most part, confined to bed and given diuretics. These accentuated at least two of the predisposing factors for thrombosis; these being stasis or slowing in the rate of venous blood flow, and changing the physical composition of the blood. It is likely that a prolongation of the life span of patients with a severe cardiac condition accounts in part for the increased number of patients with pulmonary embolism encountered yearly.

The second largest group with pulmonary embolism consisted of 160 patients with inflammatory conditions, or 31 per cent of the entire group. Included are all patients with infection, active rheumatic fever, collagen disease, thromboangitis obliterans and others. Seventy-eight deaths occurred in this group to give a mortality of 47 per cent. This is approximately equal to the mortality rates for the patients with heart disease and with malignant neoplasia. (Fig. 3B.) The patients in this group had restricted physical activity in most instances. Inflammation is known to increase the coagulability of blood [18] and to damage the venous endothelium in infected tissues. Thus, factors predisposing to thrombosis were again present.

One hundred fifty-six patients in this series were in the postoperative state. Fourteen other patients had had recent minor procedures such as cystoscopy, closed reduction of a fracture with the application of a cast, femoral catheterization, roentgenotherapy or immobilization in traction. This group comprised only thirty per cent of the entire series. The mortality was 27 per cent. Early ambulation was practiced, when possible, by all patients in this group. These patients had varied factors predisposing to venous thrombosis such as increased coagulability of blood from dehydration and other

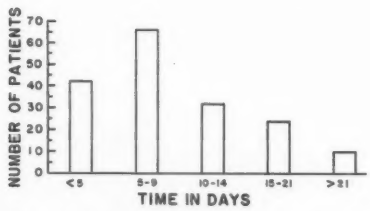


FIG. 4. Time period: procedure until embolism.

physicochemical effects of operation and anesthesia [*18*]. Restricted physical activity was imposed on the day of operation, and in spite of emphasis on ambulation practiced to some extent for a few days postoperatively.

The time from the surgical procedure to the first manifestation of thromboembolism was found to vary considerably. Most episodes occurred within the first ten days after the procedure was performed, with the highest incidence between the fifth and ninth days. (Fig. 4.)

Much has been written about the incidence of pulmonary embolism postoperatively. We have found the incidence to be low in this hospital. During the fifteen year period studied, 181,197 operative procedures, excluding deliveries, were performed in the University Hospitals of Cleveland. One hundred seventy cases of pulmonary embolism constitute a postoperative incidence of .094 per cent. Forty-two patients died postoperatively and five died after undergoing minor procedures, yielding a total mortality of .026 per cent. This is almost identical to the figure given by Fowler and Bollinger of .027 per cent [*8*], and compares favorably with a world-wide incidence of fatal postoperative pulmonary embolism of .08 per cent reported for the same period by DeBakey [*6*]. An incidence of .20 per cent has been reported by Barker et al. for an earlier period [*4*]. The incidence of pulmonary embolism, and especially fatal embolism postoperatively, has not changed greatly in the past thirty to fifty years according to these surveys. We have found the mortality from pulmonary embolism to be 5.7 per cent of the entire operative mortality for this hospital. This is in accord with the 5 to 6 per cent generally reported [*1*].

Patients with malignant neoplastic lesions are a group which is frequently discussed. Fifty-one patients, 10 per cent of this series, had cancer. The mortality for these patients was 49 per cent. Twenty-five of the patients died. Cancer in most of the patients in this group was disseminated; small localized lesions were present in only a few. Many of the patients were required to reduce their activity, many were cachectic or dehydrated and some were preterminal. The average number of patients admitted to this hospital yearly who are diagnosed as having cancer equals 3 to 4 per cent of all patients admitted. In this study the percentage of patients with cancer was about 10 per cent. However, when these patients with malignant lesions were separated from those with heart disease, or inflammation, or from those in the postoperative state, only six patients remained. This is an incidence of about 1 per cent of the 517 patients with embolism. Therefore, we believe as do others [*2*] that the association of neoplasia with thromboembolic phenomena has been overemphasized. The association is more probably on the basis of dehydration, gross tissue necrosis, cachexia and limited physical activity. There were, incidentally, only two patients in the entire series with carcinoma of the pancreas.

Finally, there are other smaller groups of patients which deserve attention. These are again found to be patients who have been required to restrict their physical activity sharply if not confined to complete bed rest. Thirty-one patients had recent cerebrovascular accidents and eighteen died, a mortality of 58 per cent. In addition to the cerebral lesion most had heart disease, often with heart failure. Twenty-eight patients were diabetic. Some patients were in chronic renal failure and were uremic. A few patients had advanced arthritis. Many in the study had chronic pulmonary diseases of varying severity such as asthma and emphysema, bronchiectasis, silicosis, silicotuberculosis, and others. In some of the patients these diseases contributed to increased pulmonary stasis and pressure and failure of the right side of the heart. Four patients had secondary polycythemia. In one a sickle cell trait was found. These five patients demonstrated the only instances of thrombogenic blood dyscrasias in the group.

MORTALITY AND CAUSE OF DEATH

The mortality in the entire series was 41 per cent. (Fig. 3B.) Analysis of the 159 autopsy reports revealed that sixty patients, or 38 per cent of the group, clearly died as a direct result of a large embolus. Fifty-four patients died

TABLE 11
CAUSE OF DEATH ASSOCIATED WITH INCIDENTAL PULMONARY EMBOLISM

Cause	Patients
Myocardial infarction	5
Overwhelming infection	3
Perforation and hemorrhage of duodenal ulcer	2
Advanced malignancy	1
Ruptured cerebral aneurysm	1
Massive cerebrovascular accident	1
Aspiration pneumonia	1
Anaphylactic reaction to penicillin	1
Renal failure and uremia	1
Iliac artery thrombosis and gangrene	1
Massive upper gastrointestinal hemorrhage	1
Total	18

within one hour. The other six patients died from two to eight hours after the embolism had occurred. In thirty-three patients this was the first embolus.

An additional eighty-one patients, 51 per cent of those autopsied, died of cardiorespiratory failure and multiple small pulmonary emboli. In these patients the emboli accentuated the cardiac failure and hastened the demise. Thus, 89 per cent of the deaths were the immediate or the delayed result of the emboli. In eighteen patients or 11 per cent, the pulmonary emboli were clearly incidental to the principal cause of death. (Table 11.)

In determining the total mortality from pulmonary embolism alone, based on the autopsied patients, we have applied this figure of 89 per cent to the entire group of 212 deaths. This would amount to 189 deaths in the total series. The over-all incidence of fatality from pulmonary embolism in the 287,370 patients would then be .066 per cent. This compares with the figures of .073 per cent found by Ochsner [*14*], and the figure of .054 per cent found by Fowler and Bollinger [*8*].

When the data are analyzed solely for the time interval between the onset of symptoms to death it is seen that one half of the entire group died within twenty-four hours of the onset of symptoms. (Fig. 5.) From a consideration of these time relationships and those figures just given for the cause of death, it is seen that the therapeutic possibilities for successful pulmonary embolectomy are limited. The most suitable patients would be those dying from a

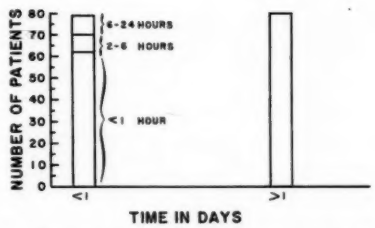


FIG. 5. Time period: onset of embolism until death.

large embolus alone, without associated heart disease but who would not die within the first hour after the onset of symptoms. This eliminates all but six patients in this series. One should exclude the patients with advanced and long-existing cardiopulmonary diseases as unlikely to benefit. A recently published case report has emphasized the potential value of this procedure in a small and highly selected group of patients [*16*].

MECHANISM OF DEATH

The mechanism of death in patients who die suddenly from pulmonary embolism is still controversial. Much experimental work has been carried out to define more clearly whether death is solely a result of mechanical obstruction of a large portion of the pulmonary arterial system or whether other mechanisms play a role. When the pulmonary arteries are suddenly blocked by emboli, acute cor pulmonale or strain of the right side of the heart ensues. Experimental studies of dogs have shown that if this blockage is greater than 70 per cent in a previously healthy cardiorespiratory system, the dog will die [*9*]. Obviously, in a diseased cardiorespiratory system, less than 70 per cent obstruction of the pulmonary arterial system will be fatal. In patients with borderline heart failure, previous chronic cor pulmonale or severely limited cardiorespiratory reserve, it is conceivable that small decreases in available pulmonary arterial circulation, occurring suddenly, may be fatal.

Because of the rapidity of death and the frequent postmortem findings of small multiple emboli, many attempts have been carried out in experiments with animals to demonstrate the existence of vasospastic reflexes, such as pulmopulmonary, pulmovagal or pulmocoronary reflexes. A recent report summarizes much of the work that has been carried out [*10*]. The evidence available to date following this experi-

mental work indicates that no such reflex mechanisms exist.

EFFECTS OF PROPHYLACTIC THERAPY AGAINST FURTHER EMBOLISM

This analysis attempts to answer the following question: having had one pulmonary embolus did prophylactic therapy with anticoagulants, especially Dicumarol, prevent further embolism or death if embolism occurred? Most studies of anticoagulant therapy have been concerned with the entire problem of thromboembolism. However, many instances of venous thrombosis occur which probably never become embolic. Therefore, we chose to study only the group of patients who had had a pulmonary embolus, rather than the entire group. It is known definitely that the potential for subsequent emboli exists in these patients.

The comparison group of patients not treated with anticoagulants or by ligation of veins included the following four groups: (1) Those patients, observed during 1943 to 1946, before anticoagulants were in general use in these hospitals for thromboembolic complications; (2) those patients, in every year, not given anticoagulant therapy because of the presence of some contraindication to anticoagulants such as gastrointestinal bleeding, cerebrovascular accident with suspected hemorrhage, and others; (3) those patients in whom the diagnosis of pulmonary embolism was not made until four to five days after its occurrence, at which time the patient had improved so satisfactorily without such therapy that it was not started; (4) those patients who received anticoagulants for less than forty-eight hours, after which time administration thereof was discontinued, who subsequently had a recurring embolus.

In this evaluation of the effects of prophylactic therapy we have used only those patients in whom pulmonary embolism was diagnosed clinically and confirmed at postmortem examination and in whom the choice existed for treatment with anticoagulants and/or ligation of veins. We have thus excluded twenty-eight patients who died but were not examined postmortem since the cause of their death could not be determined accurately. We have excluded seventy-five patients who died within forty-eight hours following embolism occurring for the first time, since it is unlikely that therapy could affect their course. We have also excluded fifty-one patients, who died in congestive failure, in whom pulmonary embolism was not suspected clinically. Finally, we have excluded the eighteen patients whose deaths were not related to their pulmonary embolism.

There were 356 patients suitable for inclusion in this analysis with a total of 385 embolic episodes. Fifty-one patients were examined post mortem. (Figure 6 summarizes the results.)

It should be noted that the number of patients in the group treated with anticoagulants and/or ligation of veins was about two and six-tenths times the number in the group treated solely by bed rest, elastic bandages and other nonspecific means. In spite of this discrepancy, there were 107 patients in the latter group, a sufficient number to make a comparison valid. There were fewer recurrent emboli in patients in the treated group as well as a mortality differential of 35.5 per cent to 4.7 per cent, a sevenfold difference.

In order to study more accurately the effect of this therapy when heart disease was present, the cases were separated into two categories; those patients with and those without heart disease. Figure 6 depicts these results. In both groups there were again patients in whom emboli recurred and a few deaths, even with the use of anticoagulants and ligation of veins. However, in the group of patients without heart disease the protective effects of anticoagulants were especially apparent. One death occurred in 123 patients, or .8 per cent. Comparison of this mortality to the 18.5 per cent for the untreated group indicates a difference of twenty-three times. Incidentally, we found only two patients, treated satisfactorily with anticoagulants, who had a second massive embolus with death occurring within twenty-four hours. One was receiving Dicumarol and the other Tromexan.® All other deaths of patients who were receiving anticoagulants were the result of multiple emboli. Thus, these methods of therapy are not infallible. However, we believe that the benefit of their use is striking.

Ligation of veins has not been analyzed separately for the following two reasons: (1) We have never been impressed with their efficacy, so that usage has been slight (only seventeen of the 517 patients in this series). (2) Except for five instances, ligation has been performed in conjunction with administration of anticoagulants. Ligation of veins has been performed on the bilateral superficial femoral

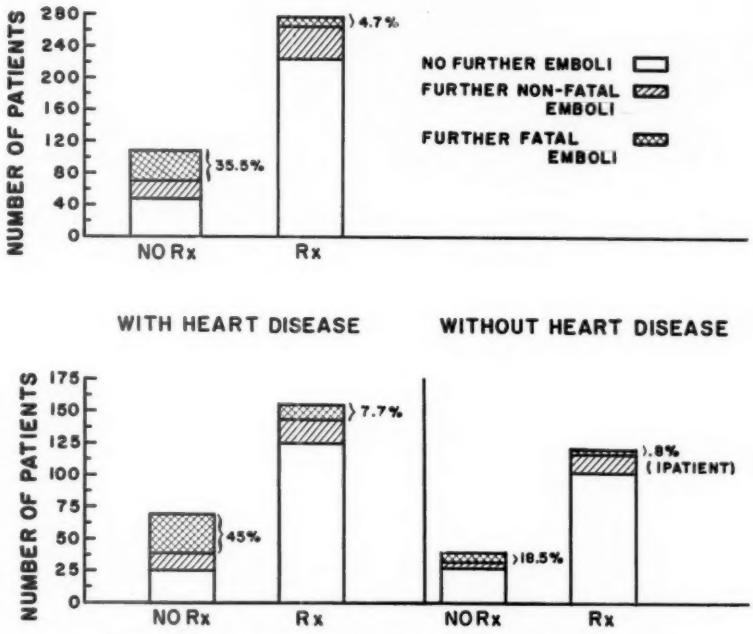


FIG. 6. Effect of prophylactic treatment. (Rx = Anticoagulants.)

veins in thirteen patients, on the great saphenous veins at the saphenofemoral bulb in two and on the inferior vena cava in two patients. In this small group there were four instances of repeated embolism, one of which resulted in death. The main problem of venous ligation is selecting the site for ligature. It is likely that if one can ligate above the thrombus, further embolism will be prevented. As observed in this study the proper site is not easily found. Only one fourth of all patients with pulmonary embolism had antecedent local symptoms or signs and a significant number of emboli arose from sources not amenable to peripheral venous ligation. Finally, we believe the results of careful anticoagulant therapy are good and have utilized it in the great majority of patients.

We have had few complications from the use of anticoagulants. One patient died of unrecognized massive gastrointestinal hemorrhage. Two patients who had been receiving anticoagulants died of infection resulting from perforation of duodenal ulcers. Associated bleeding in these two patients, however, did not appear to be the cause of death. Two patients had minor epitaxes which stopped when the blood prothrombin content was allowed to rise. One patient developed a large hematoma in a bruised area of the arm. This required drainage. Some patients developed microscopic hematuria.

In the group with vein ligations one patient had moderate edema of the ankles after superficial femoral ligations. One patient had moderate edema in the lower portion of the leg, with a stasis ulcer that required grafting twice in the year following ligation of the inferior vena cava. The other patient in whom the inferior vena cava was ligated had mild edema of the ankles only.

COMMENTS AND CONCLUSIONS

This study reveals that pulmonary embolism is a more frequent complication of medical conditions than of surgical ones. Nevertheless, because it does occur in patients who have undergone surgery, it is necessary for surgeons to have a basic knowledge of the predisposing factors, role of associated diseases, mechanisms of death, and effects of therapy. The decisions, concerning the proper time to employ venous ligation and at what site or when to attempt a pulmonary embolectomy, are difficult and must be made separately for each individual patient.

The most important factor predisposing to venous thrombosis is, we believe, interference with venous return to the heart with resultant venous stasis. Especially insidious is the relative peripheral-venous stasis brought about during hospitalization for such conditions as heart disease, especially with heart failure, inflammatory conditions, surgical procedures, neoplasia with metastases and complications, cerebrovascular accidents and uremia. That so many of these patients experience their first thromboembolic episode during a period of hospitalization attests to the fact that even such simple therapy as bed rest is not without its hazards.

The second most important predisposing factor is an increase in the coagulability of blood. Some of the factors which cause this are as follows: an increase in platelets after trauma, operation or inflammation; an increase in thromboplastic factors after injury to tissue, and an increase in coagulability from bacterial toxins [*18*]. To these must be added the sudden increase in concentration of blood constituents effected by a diuresis or by dehydration. The increased coagulability in some blood dyscrasias is not common enough to assume a large role. Finally, the element of endothelial damage plays a role, especially in inflammatory conditions and in the postoperative state.

Recognition and prevention, when possible, of these predisposing factors plus an alert awareness for early evidence of venous thrombosis must be emphasized repeatedly. The lack of a reliable diagnostic test makes one rely heavily on clinical suspicion. The use of anticoagulants should be encouraged in these situations.

On our surgical services, the program to combat thromboembolism is as follows: early ambulation and activity; close attention to position on the operating table and in bed afterwards; the avoidance of binding straps, of pillows under the knees and of tight garments around the thighs or legs; instruction to patients and nurses about active and passive leg exercises while in bed; and caution about sitting for long periods with the legs flexed. We have used elastic leg wrappings frequently, when indicated by obesity, peripheral venous stasis, cardiac failure, position on the operating table and length of operative procedure and anesthetic time. We have, in general, limited the prophylactic use of anticoagulant therapy to those patients with past histories of embolism or venous thrombosis. Administration of a dosage of heparin is initiated in these instances twenty-four to thirty-six hours following the operation. Venous ligations have not been employed on a prophylactic basis. Our primary efforts have been directed toward correcting venous stasis, which we believe is one of the more important factors in the pathogenesis of venous thrombosis. Recently others have reported preliminary efforts toward decreasing stasis in the legs during operation [*12,17*]. We have not as yet employed these methods.

The basic treatment of thromboembolic disease in these hospitals, during the fifteen year period studied, has been essentially as follows: bed rest with elevation of the lower extremities is instituted, the patient is cautioned about sudden "straining" efforts, laxatives are given as indicated, the legs are wrapped with elastic bandages if pain and tenderness are not too great and a heat cradle over the lower extremities to provide mild warmth is employed. In addition, anticoagulants are administered.

The use of anticoagulants has been somewhat variable. There is no standard routine and there is no "team" which sees all patients. Nevertheless, because of the dominant opinions of a few members of the staff most patients have received the following therapy. An immediate determination of prothrombin time is performed and 50 mg. of heparin are given intravenously and repeated every four hours. Dicumarol, 300 mg., is given orally. On the following day, 150 to 200 mg. are given, depending on the prothrombin content of the blood. Determination of the prothrombin level is repeated daily and it is maintained in the range of 10 to 30 per cent of normal by appropriate doses of Dicumarol. Heparin is discontinued after suitable prothrombin levels are achieved, usually after forty-eight hours. Bed rest is continued for about a week or until all symptoms in the leg are relieved. After two days, cautious active and passive exercises in bed are encouraged. After one week, the patient is allowed to slowly ambulate. Dicumarol is continued ideally for three weeks or until full activity is resumed.

Venous ligation is reserved for the following patients: (1) Those who continue to have emboli during good anticoagulant control; (2) those who have some contraindication to the use of anticoagulants; or (3) those who have

septic pelvic thrombophlebitis with repeated embolization. We believe there must be some indication as to the site of thrombosis so that an intelligent selection of the site of ligation may be made. To dogmatically place all ligatures at one site only, will not prevent further embolism. This point, though simple, is frequently overlooked in discussions of this subject. Suitable sites, we believe, are the superficial femoral veins when the veins in the calf, only, are involved and no thrombus is found at the site of exploration, or the inferior vena cava when veins in the thigh or pelvis are involved or when there is a thrombus in the explored superficial femoral vein. We do not advocate "sucking back" a loose clot in the femoral system found proximal to the venotomy, nor do we advocate ligation of the common femoral vein because of the deep venous insufficiency so often created.

The specific therapy of the pulmonary embolus, when it is diagnosed, is unsatisfactory, as revealed by the over-all mortality rate of 41 per cent found in this series of 517 patients. It is often the therapy of acute cor pulmonale. When the symptoms are those of severe dyspnea, cyanosis, fall in blood pressure or coma, oxygen is immediately given. If administration of anticoagulants has not previously been initiated, 50 mg. of heparin is given intravenously. In addition, many patients are given atropine intravenously in doses of .6 to 1 mg. and papaverine hydrochloride intravenously in doses of 30 mg. in spite of the lack of experimental evidence favoring the use of such agents.

In patients who have had one pulmonary embolus we have found the results of prophylactic anticoagulant therapy to be strikingly beneficial. Anticoagulants reduced the incidence of subsequent embolism from 60 per cent in the control group to 20 per cent in the treated group. Of more importance, they reduced the mortality from further embolism from 35.5 to 4.7 per cent. This reduction was even greater in those patients who had no significant cardiac disease. The mortality fell from 18.5 to .8 per cent. This reduction was essentially the result of the use of anticoagulants; ligation of veins was employed in only seventeen of the 278 patients treated and only five of these were not given anticoagulants.

More than one half of the patients who die from pulmonary embolism die from the effects of multiple small emboli in conjunction with existing cardiopulmonary diseases. Of those who die from a large or massive embolus alone, the great majority die within one hour; fifty-four of sixty patients in this series died within this period. These facts limit the successful application of pulmonary embolectomy, although in carefully selected patients it may be lifesaving.

SUMMARY

This study includes 517 patients with pulmonary embolism seen during a fifteen year period from 1943 through 1957. The incidence of pulmonary embolism during this period was .18 per cent of all patients admitted. The incidence was .49 per cent of the patients with medical disorders and only .094 per cent of the patients who were treated surgically. The primary medical condition associated with pulmonary embolism is heart disease. Sixty-five per cent of the patients in this series had significant heart disease. Eighty-one per cent of the deaths were of patients with heart disease. Postoperative patients and those with inflammatory conditions each accounted for about 30 per cent of the series. Many patients had combined diagnoses of a cardiac inflammatory condition or neoplastic disease and pulmonary embolism.

The age of the patients, sources of the emboli, location of first symptoms, the role of associated diseases, and the causes and mechanisms of death are discussed.

The results of prophylactic therapy with anticoagulants, utilizing the coumarin drugs for long term control, for the patients in this series who had a history of one diagnosed pulmonary embolus prior to therapy were strikingly beneficial. The incidence of subsequent emboli was reduced. More important, the mortality when subsequent embolism occurred was reduced sevenfold, from 35.5 to 4.7 per cent. This reduction was greater in patients without heart disease. Here, the mortality was reduced from 18.5 to 0.8 per cent, a difference of twenty-three times. We believe these findings support the continued usage of anticoagulants in the prophylaxis of pulmonary embolism.

REFERENCES

1. Allen, E. V., Barker, N. W. and Hines, E. A., Jr. Peripheral Vascular Diseases, 2nd ed., p. 524. Philadelphia, 1955. W. B. Saunders.

2. ANLYAN, W. G. and DELAUGHTER, G. D. The significance of idiopathic venous thrombosis in hidden carcinoma. *J. A. M. A.*, 161: 694, 1956.
3. ANLYAN, W. G. and HART, D. Special problems in venous thromboembolism. *Ann. Surg.*, 146: 499, 1957.
4. BARKER, N. W., MYGAARD, K. K., WALTERS, W. and PRIESTLEY, J. T. A statistical study of postoperative venous thrombosis and pulmonary embolism. *Proc. Staff Meet. Mayo Clin.* 15: 769, 1940; 16: 1, 1941.
5. BARNES, A. The problems of pulmonary embolism. *West. J. Surg.*, 1: 551, 1942.
6. DEBAKEY, M. E. A critical evaluation of the problem of thromboembolism. *Surg. Gynec. & Obst.*, 98: 1, 1954.
7. DETAKATS, G.: Management of venous thrombosis in the lower extremities. *Surgery*, 37: 507, 1955.
8. FOWLER, E. F. and BOLLINGER, J. A. Pulmonary embolism: a clinical study of ninety-seven fatal cases. *Surgery*, 36: 650, 1954.
9. HOLDEN, W. D., SHAW, B. W., CAMERON, D. B., Shea, P. J. and DAVIS, J. H. Experimental pulmonary embolism. *Surg. Gynec. & Obst.*, 88: 23, 1949.
10. KNISLEY, W. H., WALLACE, J. M., MAHALEY, M. S., SATTERWHITE, W. M. The cause of death in pulmonary embolization. *Am. Heart. J.*, 54: 483, 1957.
11. MCLACHLIN, J. and PATERSON, J. C. Some basic observations on venous thrombosis and pulmonary embolism. *Surg. Gynec. & Obst.*, 93: 1, 1951.
12. MCLACHLIN, J. and PATERSON, J. C. The peripheral venous heart. *Arch. Surg.*, 77: 568, 1958.
13. MILLER, R. and BERRY, J. B.: Pulmonary infarction; a frequently missed diagnosis. *Am. J. M. Sc.*, 222: 197, 1951.
14. OCHSNER, A., DEBAKEY, M. E., DECAMP, P. T. and DA ROCHA, R. Thromboembolism. *Ann. Surg.*, 134: 405, 1951.
15. RAVDIN, I. S. and KIRBY, C. K. Experiences with ligation and heparin in thrombo-embolic disease. *Surgery*, 29: 334, 1951.
16. STEENBURG, R. W., WARREN, R., WILSON, R. E. and RUDOLF, L. E. New look at pulmonary embolectomy. *Surg. Gynec. & Obst.*, 107: 214, 1958.
17. TICHY, V. L. Prevention of venous thrombosis and pulmonary embolism by electrical stimulation of leg muscles. *Surgery*, 26: 109, 1949.
18. WINTROBE, M. Clinical Hematology, p. 251. Philadelphia, 1951. Lea & Febiger.

Portacaval Shunt: Pre- and Postoperative Management

M. J. MACKBY, M.D., *San Francisco, California*

From the Kaiser Foundation Hospitals, San Francisco, California.

THE term portacaval shunt (as used in this article) should be interpreted as any method of surgical anastomosis between the portal and systemic venous circulation. Each type of shunt has its own particular advantages and creates its own potential postoperative hazards.

In general the preoperative preparation of the portal hypertensive patient involves the correction of various manifestations of hepatic insufficiency, mainly: (1) derangements of the clotting mechanism of the blood demonstrated by a prolonged prothrombin time; (2) impairment of the aldosterone destructive mechanism of the liver as manifested by increased renal tubular reabsorption of sodium and water; (3) inability of the liver parenchyma to complete the ornithine cycle and convert circulating blood ammonia into urea [*6,19*].

The shunt procedure should be tailored to the particular needs of the patient. The patient with cirrhosis, massive variceal bleeding, marked portal hypertension, but little or no clinical evidence of ammonia intoxication will require maximal portal decompression with secondary consideration given to possible postoperative episodic stupor. This patient usually has a well compensated cirrhosis with adequate

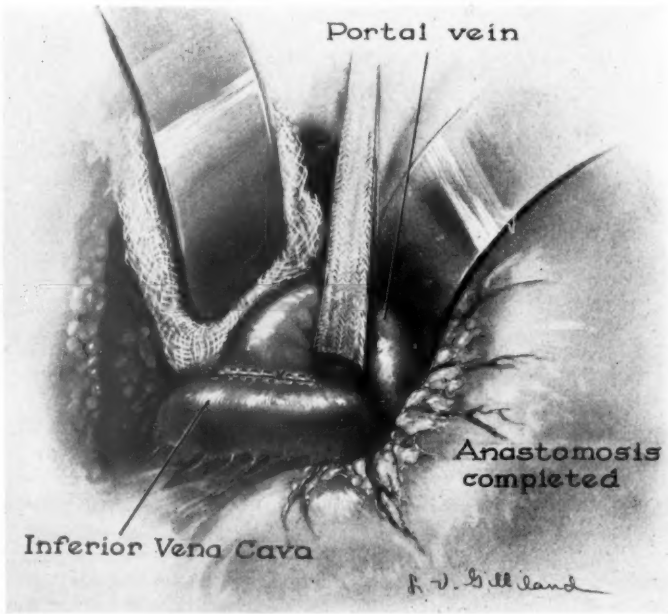


FIG. 1. End to side portacaval shunt.

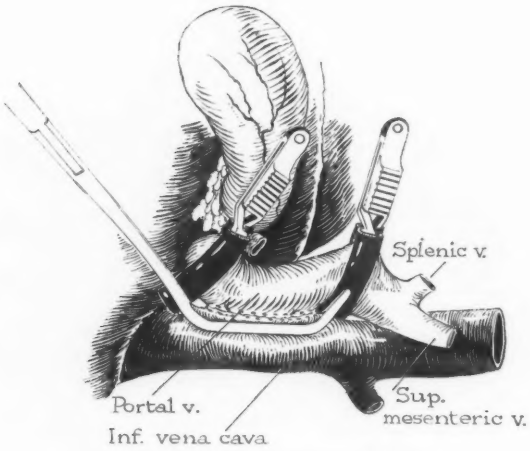


FIG. 2. Side to side portacaval shunt.

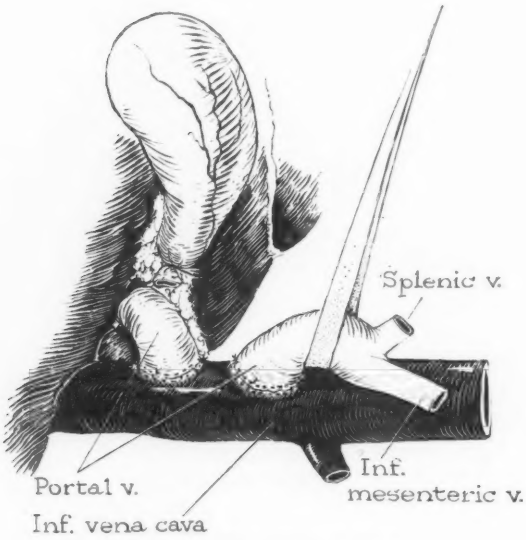


FIG. 3. Double portacaval shunt.

liver regeneration but inadequate retroperitoneal collateral circulation, and should undergo an end-to-side portacaval shunt. The end-to-side shunt (Fig. 1) affords maximal portal decompression, minimal chance of postoperative anastomotic thrombosis, and, in a patient with good hepatic reserve, minimal chance of postoperative ammonia intoxication [*17*].

The patient who has recurrent bleeding from esophageal varices and who, with each bleeding episode, becomes disoriented, agitated and has a gross flapping tremor may tolerate an end to side portacaval shunt very poorly. In this situation it may be better to perform a splenorenal shunt and accept the risk of postoperative thrombosis and/or insufficient reduction of portal pressure as a lesser evil than the production of a serious recurrent neurologic deficit.

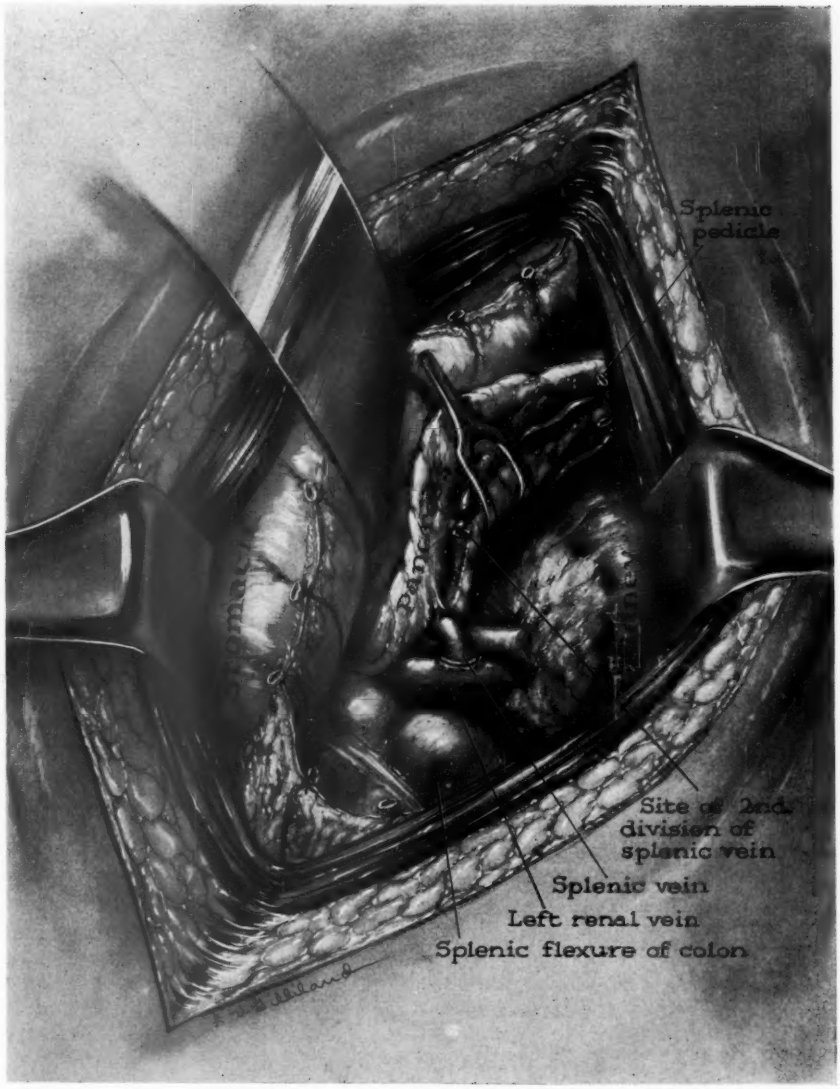


FIG. 4. Splenorenal shunt.

The patient with cirrhosis and bleeding who is also suffering from refractory ascites may receive maximum benefit from a side to side [15] (Fig. 2) or double portacaval shunt [18], as both procedures produce maximal hepatic as well as splanchnic decompression. A double shunt is preferable to a side to side shunt if the portal vein can be lengthened sufficiently to make the operation technically feasible. (Fig. 3.) In the presence of massive upper abdominal adhesions or a large caudate lobe, either operation may be difficult or impossible to perform; even when side to side shunt construction presents no obstacle, one is faced with the hazard of postoperative anastomotic occlusion due to eddy currents and turbulence produced by this method of venous communication.

Extrahepatic block developing early in life,

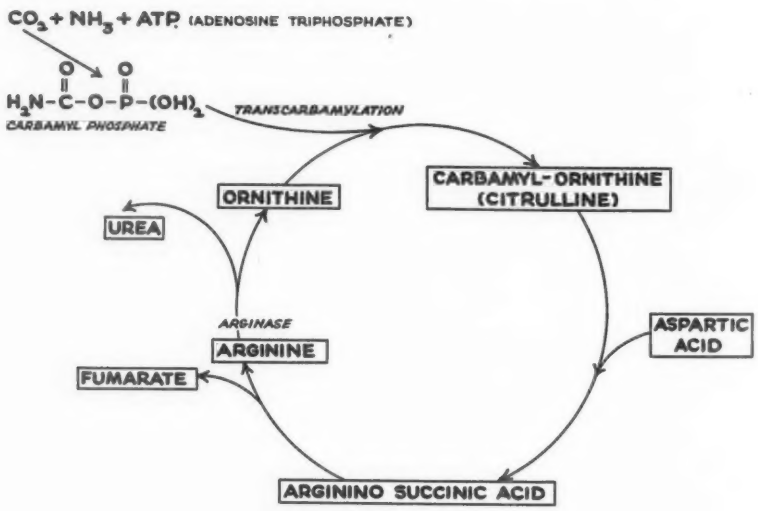


FIG. 5. The urea cycle.

possibly secondary to omphalitis and thrombosis of the portal vein presents a special problem. Liver function is good and therefore the child or young adult will tolerate surgery well. Ideally the portal hypertension and associated esophageal bleeding should be corrected by the construction of a splenorenal shunt [*14*] (Fig. 4.) However, unless the child is at least eleven or twelve years old and has associated marked splenomegaly, the splenic vein is apt to be disappointingly small. A splenic vein measuring less than 0.8 cm. in diameter as determined by preoperative percutaneous splenoportography [*2*] will be unsatisfactory for shunt construction and/or continued patency. Therefore, it is well to tide the younger child over each successive bleeding episode by means of hospitalization and massive transfusions. In such situations transthoracic ligation of the bleeding varices and even esophagogastrectomy [*26*] have been advocated although both measures are temporary as new collaterals will reform in six weeks to eighteen months; however, sufficient time may be gained by such delaying tactics to permit the splenic vein to enlarge to the point at which it can be utilized for the construction of a splenorenal shunt [*17*].

PREOPERATIVE PREPARATION

The preoperative preparation of the shunt candidate should include the following:

1. Measures to control active bleeding. This includes the active and skillful use of the Sengstaken, Blakemore or Linton tubes. The exact details of tube management have been discussed at length both by developers of the tube and myself [*5,14,17*]. Directions are included with each tube as it comes from the factory. It is obvious that the bleeding must be coming from lower esophageal or upper gastric varices and the tube must be passed over the varices gently to avoid possible increased bleeding. Once the tube is in place the patient cannot swallow and aspiration pneumonia is a real hazard. Frequent and adequate aspiration of secretions if the patient is heavily sedated (usually a necessity with balloons inflated and traction applied), or expectoration of secretions if the patient is not sedated and alert, is essential to prevent this lethal complication.

2. Measures to control preoperative ammonia intoxication. This is most apt to occur following variceal hemorrhage and to progress rapidly through the stages of confusion, gross flapping tremor, agitation, disorientation, coma and death unless the hemorrhage is controlled promptly. The offending blood is removed from the gut or neutralized by: (1) nasogastric suction and lavage; (2) colonic irrigation with 1 per cent neomycin solution; (3) the conversion of the foreign protein in the intestine to ammonia by the protein splitting action of intes-

tinal bacteria is checked by the sterilizing action of neomycin (both through the nasogastric tube and in colonic lavages); (4) the conversion of circulating blood ammonia (in the patient in hepatic decompensation) into urea, which can be excreted by the kidney, by the intravenous administration of 50 gm. of arginine in glucose [19] once, twice or three times daily. (Fig. 5.)

3 Measures to correct clotting deficiencies in the blood. The faulty production of vitamin K by the liver may be aggravated by intestinal sterilization with elimination of vitamin K production by the intestinal flora. Intramuscular injection of 72 mg. of vitamin K_1 (Hykinone®) daily may bring a disturbed clotting mechanism back to normal. In the presence of advanced hepatic insufficiency, however, administration of vitamin K_1 may still further depress prothrombin levels [25], which should therefore be checked daily. If the prothrombin level declinesc progressively with the administration of vitamin K_1 the latter should be discontinued and reliance placed upon transfusions of fresh whole blood, in plastic bags.

4. Measures to eliminate ascites. This can be accomplished by restricting sodium intake to 10 mEq. daily, by the administration of 2 cc. of a mercurial diuretic intramuscularly followed by aminophyllin several times weekly [6,10], (Diamox® and Diuril® [16] should not be administered as they are capable of producing or increasing ammonia intoxication), and by the elevation of serum albumin to as close to 3 gm. per ml. as possible by the administration of intravenous salt-poor human albumin (expensive and only of limited effectiveness).

5. Measures to increase the effectiveness of liver parenchymal function. During acute insufficiency or actual coma, the administration of nasal oxygen at a flow rate of 6 to 7 L. per minute, intravenous hypertonic glucose in a 10 per cent solution, the use of intramuscular liver extract (of debatable value) in conjunction with intravenous arginine, may be lifesaving and may bring a comatose, jaundiced, ascitic patient back to a stage of hepatic compensation during which he can qualify for shunt construction.

6. Proper timing of surgery. Shunt surgery performed during or immediately after acute massive upper gastrointestinal bleeding carries a forbidding mortality of at least 30 per cent. In institutions which do not handle such problems routinely and in which the nursing, surgical and anesthesia staffs are not fully cognizant of the complex physiologic problems involved, the mortality consequent to emergency shunt surgery may be much higher. The operative management of acute bleeding varices is not within the scope of this discussion, but it is interesting to note in this connection that leading authorities disagree markedly in their recommendations. My experience with either definitive shunt surgery or ligation of varices during acute bleeding episodes has not been felicitous. Accordingly I rely upon the Sengstaken tube to control the acute bleeding episode. A month is not too long to allow for replenishment of blood depots and hepatic regeneration following massive hemorrhage into the intestinal tract. When the liver function tests have shown a maximum degree of improvement and level off (usually in four to six weeks), surgery is then undertaken provided the patient is not jaundiced, has a clear sensorium, a minimum of flapping tremor, and a minimal degree of ascites.

If the Sengstaken tube fails to control bleeding, or if bleeding recurs upon deflation of the balloons in forty-eight hours, direct ligation of the varices should be performed transthoracically in very obese patients or in those in whom the diagnosis is certain; otherwise it should be performed transabdominally [17]. Six weeks following this procedure a definitive shunt should be performed. If the patient is allowed to go much longer than this period without portal decompression, rebleeding may occur from new varices [14].

POSTOPERATIVE MANAGEMENT

Postoperative care of the shunt patient should include the following:

1. Measures to protect the liver parenchyma (usually not necessary in the patient with extrahepatic block and good liver function following splenorenal shunt construction). Any general anesthetic agent and most preoperative sedatives must be detoxified by the liver and some are actively hepatotoxic. Anoxia during induction, surgery or recovery [23], combined with surgical destruction of tissue place additional severe strain upon an already damaged liver and may produce acute hepatic decompensation in the immediate postoperative period. To afford maximal protection to the liver during the crucial first four or five days postopera-

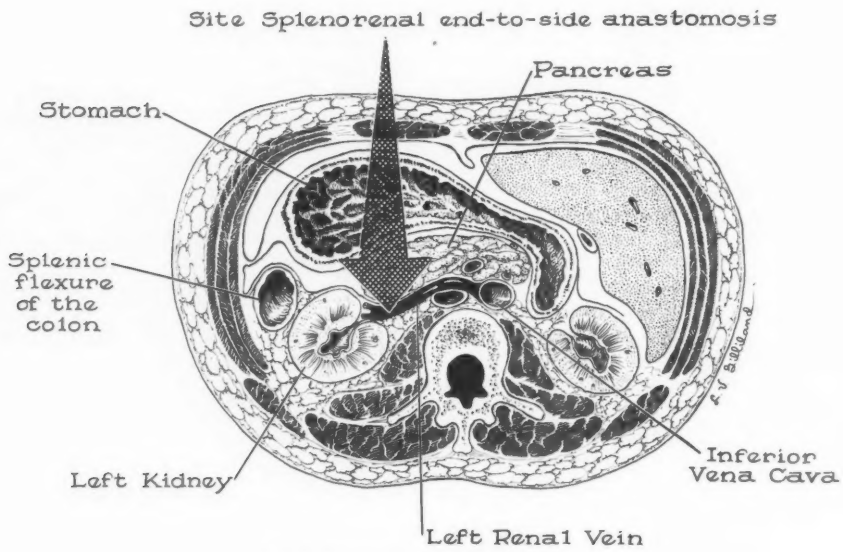


FIG. 6. Compression of left renal vein.

tively, intranasal oxygen is administered continuously at a flow rate of 6 to 7 L. per minute, and intravenous 10 per cent glucose in distilled water is given to supply nutriment to the parenchymal cells. Any anemia is corrected by the use of fresh whole blood in plastic containers (banked blood is deficient in platelets, many of the red cells are hemolyzed, and if stored for as long as three weeks each unit may contain as much as 3,000 gammas of ammonia, an amount sufficient to produce hepatic coma in a patient with advanced cirrhosis).

The patient who has been subjected to an end to side portacaval shunt and has manifested any clinical signs of ammonia intoxication preoperatively even of minor degree (such as a flapping tremor or a sensation of "fuzziness in the head") should be treated vigorously postoperatively, with daily intravenous administration of 50 gm. arginine at least once and as often as two or three times daily if confused, flapping, disoriented or stuporous [19]. On a long range basis these patients should be treated with a high-carbohydrate reduced-protein (40 to 60 gm.) diet, plus 200 mg. of neomycin and 20,000 units of bacitracin daily [5]. This minimal antibiotic dosage appears to reduce the bacterial flora of the intestine sufficiently to keep the ammonia level of the circulating blood within asymptomatic clinical limits. Two objections to this regimen are: (1) the considerable expense involved ($90.00 a month at current prices), and (2) some interference with vitamin K production by reduction of the normal intestinal flora.

The construction of a side to side portacaval or splenorenal shunt is followed by an early or delayed incidence of anastomotic thrombosis four to nine times as great as that subsequent to end to side portacaval shunt. Increasing abdominal distention, rising temperature (without some other obvious cause), weakness and lassitude, and vomiting of blood or coffee ground material are all ominous signs of impending anastomotic occlusion four to seven days after shunt construction and should be treated promptly and vigorously at onset with anticoagulant therapy (heparin and dicumarol).

Following the establishment of a splenorenal anastomosis it must be kept in mind that the left renal vein is longer than the right, passes over the vertebral column and is compressed between it and the overlying distended viscera in the immediate postoperative period, partially compressing the vein lumen and reducing the velocity of blood flow past the anastomic site (or sites in the case of a vein graft). (Fig. 6.) In a low pressure system with a narrow pressure differential between the splenic and renal veins and a narrow anastomotic aperture, the risk of

thrombosis is a real one, and added factors impeding blood flow such as intestinal distention may be sufficient to produce an unsuccessful result. Therefore strenuous efforts should be made to maintain intestinal decompression postoperatively by the use of a nasogastric tube with continuous suction, colonic irrigations and the administration of Prostigmin® if necessary to combat ileus. Anticoagulant therapy should be considered and employed in equivocal situations much more frequently than in end to side portacaval shunt. Objective methods of determination of shunt patency should be employed in any doubtful situation.

Disappearance of esophageal varices on esophagogram is evidence suggestive of shunt patency [*22*]. Ether may be instilled into the rectum or duodenum. Detection of ether odor in the breath of the patient occurs more rapidly in the patient with a patent shunt [*3*]. The rectum-to-lung transit time is less than thirty seconds in the presence of a patent shunt, as compared with over a minute in the patient with an occluded shunt. In an analogous test 100 mc. of sodium^{24} can be introduced into the rectum, and the time necessary for this substance to reach a brachial artery is determined with a Geiger counter over the antecubital fossa [*8*]. Over a period of ten minutes 150 mg. per kilo of sodium cholate may be instilled through a duodenal tube; in the presence of a patent portacaval shunt the bile salt level will remain persistently elevated in the peripheral blood stream [*1*]. Increased plasma volume in patients with cirrhosis will usually return to normal and the bromsulphalein level will fall significantly after successful portacaval anastomosis. Postoperative hepatic wedged pressure determinations are apt to be still further elevated following end to side portacaval shunt, as the portal system is no longer continuous with the hepatic circulation and therefore reduced pressure in the portal system is no longer reflected in the hepatic circulation [*24*]. Persistently elevated hepatic sinusoidal pressure is simply a reflection of heightened arterial tension, presumably through intrahepatic arteriovenous shunts in cirrhosis. On the other hand, with the creation of a side to side portacaval or splenorenal shunt, hepatic wedged pressure determinations should be of distinct value as reflecting the extent of postoperative portal decompression and thus the patency and efficiency of the surgical anastomosis.

The implantation of a fine gauge polyethylene catheter [*7*] in the high pressure portal side of the portacaval anastomosis has been employed in order to administer a continuous heparin drip. As the hazard of thrombosis is relatively small in end to side portacaval shunt construction, this precaution was soon found to be unnecessary and even somewhat disadvantageous as occasional seepage of blood through the vessel wall around the catheter or at the suture line due to the continuous administration of heparin may cause an occasionally serious or even fatal hematoma. However, in the construction of a side to side portacaval or splenorenal shunt, or in the rare procedure in which one of the tributaries of the portal system must be utilized for decompression, the implantation of a polyethylene catheter at some point in the portal circulation may be useful not only as a direct method of continuous heparin administration over the suture line but also for obtaining direct and repeated portal pressure determinations.

Troublesome ascites preoperatively may be aggravated postoperatively by conventional end to side portacaval shunt. Abdominal paracentesis either pre- or postoperatively should be avoided except as a last-ditch measure to reduce respiratory or digestive embarrassment [*6*]. Repeated abdominal paracentesis stimulates still greater production of ascitic fluid, depletes the body of valuable albumin and electrolytes and may cause inadvertent intestinal perforation or chronic peritonitis. The measures outlined to combat ascites preoperatively may be inadequate at that time and even more so postoperatively. Double portacaval shunt in limited clinical trial seems to afford promise as a method of controlling unrelenting ascites [*18*] but presents serious technical problems at surgery in many patients. Amphenone [*27*], a synthetic preparation which counteracts renal tubular reabsorption of sodium and water (presumed to be due to excess circulating aldosterone), is too toxic to be used safely. Currently other aldosterone antagonists are being investigated.* Bilateral adrenalectomy is one solution that has been suggested [*11*], but if the resulting adrenal insufficiency is completely controlled medically, the ascites recurs.

A word of caution is indicated regarding the rigorous restriction of salt intake in the treat-

* Spirolactone (400 mg. daily) appears to be the best of these preparations.

ment of ascites either pre- or postoperatively. It is possible to produce an acute postoperative hyponatremic episode with vascular collapse and a fatal outcome, due to an unduly severe and prolonged preoperative dehydrating regimen [6]. Following any shunt procedure, and particularly after portacaval shunt, a daily electrolyte panel and prothrombin time are essential in the intelligent management of incipient electrolyte imbalance. A drop in the serum sodium level below 120 mEq. per ml. with a concomitant rise in serum potassium should be counteracted vigorously by the administration of 5 per cent saline solution intravenously as physiologic salt solutions are not adequate to correct this dangerous situation.

COMMENTS AND CONCLUSIONS

Portacaval shunt in one of its variations is primarily a procedure to combat the most lethal complication of portal hypertension, namely recurrent hemorrhage from esophageal varices. Unfortunately, the most efficient and dependable method of portal decompression, end to side portacaval shunt, is not possible in extrahepatic block and, when performed on a patient with a sensitive central nervous system, is capable of producing or aggravating the syndrome of ammonia intoxication.

Alternative procedures such as side to side portacaval and splenorenal shunts are less apt to produce ammonia intoxication; however they are less efficient as decompressive measures and far more prone to develop anastomotic occlusion than end to side portacaval shunt. The reasons for this and methods of diagnosis and treatment of anastomotic thrombosis are detailed.

Ascites as a complication of cirrhosis either with or without recurrent esophageal bleeding can be a particularly refractory condition. The enormous variety of surgical procedures which have been advocated since 1886 and the constant development of new ones would suggest that this is a physiologic disturbance which is not readily amenable to mechanical correction. Medical management is frequently ineffective and, when combined with repeated albumin leaching, abdominal paracentesis may be actually detrimental. Surgical procedures which seem to affect ascites favorably include end to side portacaval shunt (which frequently although transiently aggravates the ascites because of immediate postoperative liver damage), double portacaval shunt and several other procedures (which are outside the scope of this discussion but will be mentioned for the sake of completeness), namely, bilateral adrenalectomy [11], ileoentectropy [20] and supradiaphragmatic transposition of the spleen [21]. In evaluating the candidate for surgical correction of ascites it must be borne in mind that ascites is an expression of advanced liver disease and that any operative procedure will be attended by a high mortality and a high incidence of postoperative complications. Wound infections, leakage of ascitic fluid, peritonitis, dehiscence and all the manifestations of acute hepatic insufficiency, namely, disorientation, coma, jaundice and gross derangements of the clotting mechanism causing hemorrhage from the operative site or from the gastrointestinal tract, are common. In evaluating the results of medical or surgical therapy one must remember that at least 7 per cent of all cases of ascites due to cirrhosis will disappear spontaneously in the absence of any treatment.

REFERENCES

1. Akita, H., Kuck, J. F. R., Walker, G. L. and Johnson, C. G. Further application of the enterohepatic circulation of bile acids to a study of the patency of a portacaval shunt. *Surgery*, 39: 230, 1956.
2. Abeatici, S., Campi, L. and Ferraro, R. Transparietal splenoportography; clinical value and indications. *Minerva med.*, 1: 1370, 1952.
3. de Almeida, A. D. Ether test in evaluation of permeability of direct anastomosis between portal and caval systems: preliminary report. *Rev. med. e cir. São Paulo*, 26: 105, 1950.
4. Blakemore, A. H. Portacaval shunt for portal hypertension. *Mod. Med.*, 26: 105, 1958.
5. Blakemore, A. G. and Voorhees, A. B., Jr. Nutrition requirements and management in patients with cirrhosis pre- and postoperatively. *Ann. Surg.*, 147: 875, 1958.
6. Blank, J. H. Clinical Recognition and Management of Disturbances of Body Fluids. Philadelphia, 1956. W. B. Saunders Co.
7. Castenfors, H. and Nordenstroem, B. Postoperative control of portal circulation with indwelling catheter in portal vein. *Acta chir. scandinav.*, 110: 464, 1955.
8. Deterling, R. A., Powers, S. R. and Bhonslay, J. B. The use of radioactive sodium in the determination of patency of portacaval shunts. *S. Forum*, 5: 193, 1954.
9. Drapanas, T., Becker, D. R., Schenck, W. G., Jr., Shaw, W. W., Potter, W. G. and Stewart, J. D. Ammonia intoxication from portal diversion and hepatic failure. *Ann. Surg.*, 142: 560, 1955.
10. Faloon, W. W., Eckhardt, R. D., Cooper, A. M. and Davidson, C. S. The effect of human serum

albumin, mercurial diuretics and a low sodium diet on sodium excretion in patients with cirrhosis of the liver. *J. Clin. Invest.*, 28: 595, 1949.

11. Giuseffi, J., Werk, E. E., Jr., Larson, P. U., Schiff, L. and Elliot, D. W. Effect of bilateral adrenalectomy in a patient with massive ascites and post-necrotic cirrhosis. *New England J. Med.*, 257: 796, 1957.
12. Hallenbeck, G. A. and Shocket, E. An evaluation of portacaval shunts for portal hypertension. *Surg. Gynec. & Obst.*, 105: 49, 1957.
13. Kark, R. M., Keeton, R. W., Calloway, N. O., Morey, G. R., Chapman, R. A., and Kyle, R. H. A rational basis for the use of low sodium, high protein diet therapy in Laennec's cirrhosis. *Arch. Int. Med.*, 88: 61, 1951.
14. Linton, R. R. In: Schiff, L. Diseases of the Liver, pp. 195–227. Philadelphia, 1956. J. B. Lippincott.
15. Longmire, W. P., Jr., Mulder, D. G., Mahoney, P. S. and Mellinkoff, S. W. Side-to-side portacaval anastomosis for portal hypertension. *Ann. Surg.*, 147: 881, 1958.
16. Mackie, J. E., Stormont, J. M., Hollister, R. M. and Davidson, C. J. Production of impending coma by chlorothiazide and its prevention by antibiotics. *Clin. Res.*, 6: 301, 1953.
17. Mackby, M. J. The Surgical Treatment of Portal Hypertension, Bleeding Esophageal Varices and Ascites. Springfield, Ill. Charles C Thomas. (In publication.)
18. McDermott, W. V., Jr. The treatment of cirrhotic ascites by combined hepatic and portal decompression. *New England J. Med.*, 259: 897, 1958.
19. Najarian, J. S. and Harper, H. A. A clinical study of the effect of arginine on blood ammonia. *Am. J. Med.*, 21: 832, 1956.
20. Neumann, C. G., Adie, G. C. and Hinton, J. W. The absorption of ascitic fluid by means of ileoentectropy in patients with advanced cirrhosis. *Ann. Surg.*, 146: 700, 1957.
21. Nylander, P. E. A. and Turunen, M. Transposition of the spleen into the thoracic cavity in cases of portal hypertension. *Ann. Surg.*, 142: 954, 1955.
22. Palmer, E. D. The fate of esophageal varices in cirrhosis following surgical portal decompression. *Gastroenterology*, 32: 861, 1957.
23. Rappaport, A. M., MacDonald, M. H. and Borowy, Z. J. Hepatic coma following ischemia of the liver. *Surg. Gynec. & Obst.*, 97: 748, 1953.
24. Redeker, A. G., Geller, H. M. and Reynolds, T. B. Hepatic wedge pressure, blood flow, vascular resistance and oxygen consumption in cirrhosis before and after end-to-side portacaval shunt. *J. Clin. Invest.*, 37: 606, 1958.
25. Rousselot, L. M. In: Liebowitz, H. R. Bleeding Esophageal Varices, Portal Hypertension, pp. 848–944. Springfield, Ill., 1959. Charles C Thomas.
26. Shafer, P. W. and Kittle, C. F. Partial esophagogastrectomy in the treatment of esophagogastric varices. *Arch. Surg.*, 61: 235, 1950.
27. Summerskill, W. H. J. and Crabbé, J. Effect of amphenone therapy on urinary excretion of aldosterone and sodium in hepatic cirrhosis with ascites. *Lancet*, 2: 1091, 1957.

Further Observations on the Surgical Treatment of the Dumping Syndrome

DAVID B. HINSHAW, M.D., CLARENCE E. STAFFORD, M.D. AND EUGENE J. JOERGENSON, M.D.,
Los Angeles, California

This work was supported by Grant No. A-2,329 of the National Institutes of Health; the Department of Surgery, College of Medical Evangelists, School of Medicine; the Los Angeles County General Hospital and the White Memorial Hospital.

THE most important undesirable feature which sometimes follows partial gastrectomy is the symptom complex known as the "dumping syndrome." It appears promptly after eating, is chiefly characterized by nausea, weakness, sweating, palpation, varying degrees of syncope, often a sensation of warmth and sometimes by diarrhea, and is usually improved by lying down. It has been well established during recent years by various investigators that there are definite physiologic changes which occur during these dumping attacks; among these are a fall in plasma volume, electrocardiographic and electroencephalographic changes, and disturbances in peripheral blood flow [*4,5,9*]. It has been observed that people who have severe dumping symptoms are probably those who have abnormal or exaggerated vascular homeostatic responses to sudden, sharp reductions in plasma volume. It has also been postulated that the vasomotor symptoms and also the electrocardiographic and electroencephalographic changes in these patients might be associated with decreased coronary and cerebral blood flow [*3,4*]. Current studies in dogs, in which "simulated dumping" was induced by injections of hypertonic glucose into the upper intestine, showed a definite increase in mesenteric blood flow which was in turn associated with a simultaneous reduction in carotid and renal blood flow. These changes in visceral blood flow were accompanied by a significant fall in cardiac output. These findings strongly suggest that there is a redistribution of the cardiac output under these circumstances which tends to deprive temporarily certain vital visceral areas [*7*]. Mathews and coworkers [*8*] have recently shown that the decrease in effective circulating blood volume in the dumping syndrome (in dogs) is probably greater than first realized. They also suggest that this probably indicates "blood pooling" or some significant redistribution of blood flow.

Most investigators agree that severe clinical dumping is closely related to very rapid gastric emptying and to the amount of stomach removed at operation. Actually, dumping symptoms may follow any operation in which the pyloric sphincter mechanism is disturbed. Although no type of gastric operation is entirely immune from this problem, there is at least some evidence to suggest that the incidence of severe dumping is probably less following the Billroth I modification than following the Billroth II reconstruction. There is also much debate as to whether the size of the gastroenteric stoma significantly affects the emptying rate of the gastric remnant. Porter and Claman made observations on 118 patients and their findings suggest that a small (2.5 cm.) stoma is definitely preferable. Dragstedt [*1*] also has noted a decrease in the incidence of dumping symptoms with small sized gastrojejunostomies. Eiseman [*10*] and his associates recently performed a controlled experiment on stomal size in dogs and concluded that there was no significant difference in emptying time between the large and the small stoma. Using a different method, Syphers and Hinshaw [*11*] also found no significant difference in the emptying rate between large and small stomas in dogs. They also noted that the small stoma was frequently more efficient as an emptying mechanism than

was the large. It is difficult to know how much of these data drawn from experiments with dogs can be transferred to the human clinical situation.

Among the surgical procedures sometimes used for patients with severe dumping symptoms are simply a narrowing of the gastrojejunostomy stomas or changing them to gastroduodenal reconstructions with or without decreasing the size of the stoma itself. Converting Billroth II operations to Billroth I reconstructions in patients who have dumping problems has been advocated by several surgeons. The chief purpose of this paper is to present a follow-up report on five patients previously treated for severe dumping by changing a large stoma Billroth II operation to a small stoma end-to-side gastroduodenostomy (a modified Finney-Von Haberer operation) [5], and to make observations on eight additional patients subsequently treated by the same method.

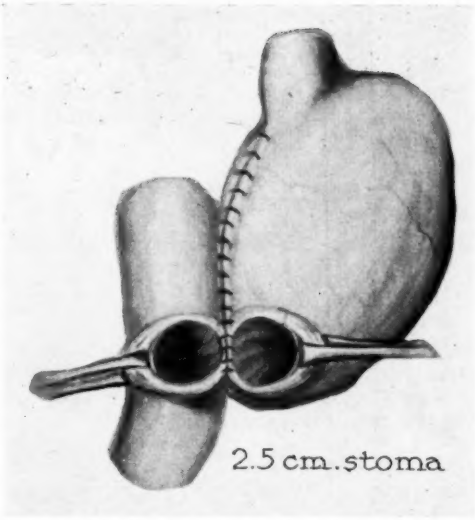


FIG. 1. This illustrates the end to side, small stoma gastroduodenal anastamosis used for operative revision in these patients.

METHODS AND PROCEDURES

All thirteen patients in this series had severe dumping symptoms following a large stoma Billroth II gastric resection without vagotomy. In each instance this surgical arrangement was changed to a Billroth I end to side (Finney-Von Haberer) type reconstruction modified by the use of a small stoma, as illustrated in Figure 1. All patients were identified as "dumpers" by severe reactions to oral test meals of 150 cc. of 50 per cent glucose solution as well as by clinical history. In ten of the thirteen cases it was possible to obtain satisfactory plasma volume and peripheral blood flow studies as well. Identical follow-up studies were also possible in ten of the thirteen; whereas, the other three were followed up by clinical history alone. The plasma volume determinations were made using Gibson's indirect modification of the Evans blue dye method, and the peripheral blood flow and skin temperature measurements were obtained with the Windsor pneumophlethesmograph and from thermisters attached to suitable digits.

RESULTS

Eight of the thirteen patients had marked clinical improvement after the converting operation. In six of these it was possible to obtain both pre- and postoperative measurements of plasma volume and blood flow which showed definite objective improvement. In four of the thirteen patients moderate clinical improvement was observed, and the objective physiologic studies in these patients roughly paralleled in magnitude the subjective improvement. One patient showed only slight clinical improvement, and it is interesting to note that there was no significant difference between the results of pre- and postoperative physiologic studies in this person. In those patients experiencing marked or moderate improvement there was persistent weight gain and the dumping attacks were either completely stopped or at least decreased in number and intensity. Twelve of the thirteen patients who were operated on considered themselves to have good or excellent results.

The first five patients in the group, who were previously reported in more detail, have now been followed up for a period of more than four years. All of these patients have substantially maintained the improvement which occurred immediately after their operative procedures. One patient who had long term follow-up study as well as a patient who was operated on more recently are subsequently considered in more detail.

CASE REPORTS

CASE 1. B. L. S., a fifty year old woman, underwent conversion of a large stoma Billroth II gastric

TABLE I
SUMMARY OF PHYSIOLOGIC VALUES BEFORE AND AFTER SURGICAL REVISION IN CASE I

Physiologic Values	During Induced "Dumping Attack" Preoperatively	Six Wk. after Surgical Revision	Four Yr. after Surgical Revision
Plasma volume (% change)	29% decrease	10% decrease	Undetermined
Peripheral blood flow	Tenfold increase	Twofold increase	Threefold increase
Clinical reaction	Severe	Mild	Mild

resection to a small stoma end to side Billroth I arrangement over four years ago, because of severe incapacitating dumping symptoms. Preoperative observations made following the ingestion of a glucose test meal showed significant changes in plasma volume, peripheral blood flow and skin temperature as well as in severe dumping symptoms. Identical follow-up studies were performed two months after the surgical revision and again four years later. There was a definite reversion of the abnormal preoperative findings toward normal in each of these follow-up studies. This patient had significant relief from her previously incapacitating dumping symptoms. She has maintained essentially all her improvement since her revision operation. She weighs 20 pounds more than preoperatively and has only occasional dumping attacks which tend to be milder than they were previously. Table I is a summary of the physiologic values and clinical reaction before and after surgical revision in this patient.

CASE II. E. L., a fifty year old man underwent conversion of a large stoma Billroth II gastric resection to a small stoma end to side Billroth I procedure over ten months ago, because of a severe dumping syndrome. Observations made before revision following a glucose test meal showed pronounced changes in plasma volume, peripheral blood flow and skin temperature. A severe clinical dumping reaction was also noted. Similar follow-up studies ten months later showed a favorable clinical reaction to the test meal and a definite reversion toward normal of the various physiologic values. This patient gained 20 pounds after surgical revision and has a minimum of dumping symptoms. Table II is a summary of the physiologic values and clinical reactions before and after surgical revision in Case II.

TABLE II
SUMMARY OF PHYSIOLOGIC VALUES BEFORE AND AFTER SURGICAL REVISION IN CASE II

Physiologic Value	During Induced "Dumping Attack" Preoperatively	Nine Mo. after Surgical Revision
Plasma volume (% change)	22% decrease	8% decrease
Peripheral blood flow	Ninefold increase	Twofold increase
Clinical reaction	Severe	Mild

COMMENTS

While many have the clinical impression that a small stoma is associated with slower gastric emptying, it has not been possible to substantiate this by experimental work in animals. However, the lack of confirmation from the laboratory does not necessarily nullify this clinical impression. It is possible that other mechanisms, such as certain gastroenteric reflexes, may be more important than the size of the stoma. It is well known, for example, that the presence of acid in the duodenum causes an immediate reflex inhibition of gastric peristalsis. Hunt [6] has postulated that "osmo receptors" are present in the duodenum which, when stimulated by hypertonic materials, cause reflex inhibition of gastric emptying. It has also been shown that the injection of hypertonic glucose directly into the duodenum of the dog frequently results in prompt inhibition of gastric peristalsis [11]. It is not yet clear whether this mechanism is common to all the upper small intestine or is confined to the duodenum.

In this series of patients generally good clinical results were achieved from the use of a procedure which re-established gastroduodenal continuity and narrowed the gastroenteric stoma. In several cases definite objective physiologic evidence of improvement was present in addition to clinical observation. It is difficult to determine just how much improvement in these patients was due to re-establishing gastroduodenal continuity and how much was due to changing the size of the stoma. Nevertheless, the relief of symptoms was definitely thought to be associated with physiologic changes rather than with some psychologic adjustment on the part of the patient. The reports of Wallensten [12] and others strongly suggest that the use of gastroduodenal continuity alone is a significant factor in keeping severe dumping symptoms to a minimum, particularly, if a large amount of stomach is not removed. It is

also true that while many surgeons have made no attempt to keep gastroduodenal stomas small, such anastomoses usually tend to be smaller than the average gastrojejunostomy. Henley [2] has described a gastric replacement operation in which a segment of jejunum is interposed between the gastric remnant and the duodenum in order to maintain gastroduodenal continuity. This interposed jejunal segment is thought to act as a sphincter regulating gastric emptying of the remnant. Henley reports excellent results with this operation in the treatment of patients with dumping syndromes and nutritional deficiencies. Although it is a reasonably formidable operative procedure, it may have promise in some cases.

The brief history of a patient treated by one of us (D. B. H.) is interesting with regard to continuity and stomal size. This woman underwent a 70 per cent gastric resection with end to end gastroduodenostomy for a benign gastric ulcer. Two years later what was thought to be a marginal ulcer developed, but none was found at surgical exploration. At this operation, closure of an exploratory incision in the area of the old anastamosis resulted in stenosis. To relieve this the operation was eventually changed to a large stoma Billroth II. Severe dumping symptoms promptly developed which had not been present previously.

The majority of patients in this series obtained distinct benefit from the change of the large stoma Billroth II to a small stoma end to side Billroth I procedure, and there was no mortality or significant morbidity associated with these operative changes. In view of these observations, when patients have severe, persistent, incapacitating dumping symptoms following large stoma Billroth II gastric resections, consideration should be given to converting this reconstruction to a small stoma end to side Billroth I modification.

SUMMARY AND CONCLUSIONS

1. Certain features of the pathologic physiology of the dumping syndrome are briefly discussed.

2. Patients with severe dumping symptoms following large stoma Billroth II operations have been treated by conversion of this operation to a small stoma end to side Billroth I modification. Favorable results were obtained in twelve of thirteen patients. A long term follow-up report is made on five patients.

3. Clinical and experimental data are reviewed regarding the influence of small stomas and gastroduodenal continuity on the occurrence of dumping symptoms.

4. When severe dumping symptoms occur in patients with a large stoma, Billroth II gastric resections, conversion to a small stoma, an end to side Billroth I arrangement should be considered.

REFERENCES

1. Dragstedt, L. R. Present status of vagotomy. *Illinois M. J.*, 106: 175, 1954.
2. Henley, F. A. Gastrectomy with replacement. *Ann. Roy. Coll. Surgeons, England*, 13: 141, 1953.
3. Hinshaw, D. B., Joergenson, E. J. and Stafford, C. E. Preoperative dumping studies in peptic ulcer patients. *Arch. Surg.*, 80: 738, 1960.
4. Hinshaw, D. B., Joergenson, E. J., Davis, H. A. and Stafford, C. E. Peripheral blood flow and blood volume studies in the dumping syndrome. *Arcb. Surg.*, 74: 686, 1957.
5. Hinshaw, D. B., Stafford, C. E. and Joergenson, E. J. Surgical treatment of the dumping syndrome. *Am. J. Surg.*, 94: 242, 1957.
6. Hunt, J. N., MacDonald, I. and Spurrell, W. R. Gastric response to pectin meals of high osmotic pressure. *J. Physiol.*, 115: 185, 1951.
7. Huse, W. M. and Hinshaw, D. B. Blood flow studies in the experimental dumping syndrome. *S. Forum*, 11: 316, 1960.
8. Mathews, D. H., Lawrence, W., Jr., Poppell, W., Vonamee, P. and Randall, H. T. Change in effective circulating volume during experimental dumping syndrome. *Surgery*, 48: 185, 1960.
9. Roberts, K. E., Randall, H. T., Farr, H. W., Kidwell, A. P., McNeer, G. P. and Pack, G. T. Cardiovascular and blood volume alterations resulting from intrajejunal administration of hypertonic solutions to gastrectomized patients. *Ann. Surg.*, 140: 631, 1954.
10. Silen, W., Eiseman, B. and Brown, W. H., Jr. The role of stomal size in the postgastrectomy dumping syndrome. *S. Forum*, 9: 464, 1958.
11. Syphers, C. E. and Hinshaw, D. B. Unpublished data.
12. Wallensten, S. Results of the surgical treatment of peptic ulcer by partial gastrectomy according to Billroth I and II methods. *Acta chir. Scandinav. (suppl.)*, 1954.

Epithelioma of the Tongue

RESULTS OF TREATMENT IN 217 CASES

HARVEY LASH, M.D. AND JOHN B. ERICH, M.D., *Rochester, Minnesota*

From the Section of Plastic Surgery, Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

A REVIEW of the literature on carcinoma of the tongue reveals many conflicting opinions concerning the importance of the grade of malignancy, the significance of cervical metastasis, the value of various methods of treatment and the role of prophylactic procedures. In the hope of resolving some of the confusion in our minds on this subject, we have analyzed a series of our own patients and have attempted to compare these findings with data compiled from the available world literature, which to date includes 10,454 cases of lingual cancer [*1–6,9,10,14–31*].

Data for this report were derived from the case histories and the tissue removed from all patients with squamous cell epithelioma of the tongue seen at the Mayo Clinic from 1949 through 1953. Of the 330 cases thus found, 113 were eliminated for the following reasons: examination or treatment had been started prior to 1949 in thirty-nine cases; microscopic proof was not available in ten, the carcinoma had originated in adjacent tissues and subsequently spread to the tongue in thirty-eight; the lesion was metastatic from a carcinoma primary in the lung in one, review of the tissues revealed that the original diagnosis of squamous cell epithelioma was erroneous in four, and the growth was a malignant tumor other than squamous cell epithelioma in twenty-one. Elimination of these 113 cases left a total of 217 cases of squamous cell epithelioma of the tongue.

The clinical data in these 217 cases have been tabulated. In each, the tissue was re-examined and in some cases more microscopic sections were prepared so that the pathologic diagnosis would be indisputable.

INCIDENCE

Uniform agreement exists on the preponderance of squamous cell epithelioma over other forms of malignant neoplasms involving the tongue. Epitheliomas usually are reported as constituting 90 to 95 per cent of all lingual cancers. Estimates of the frequency of this lesion as compared to that of malignant tumors elsewhere in the body vary from 1 to 4.8 per cent in the United States to as much as 14 per cent in India. Perhaps the most reliable statistics are those of the United States Public Health Service [*12*], which give the incidence as 0.8 per cent of all new cases of cancer. Mortality statistics are probably more reliable than those reflecting the incidence, and lingual cancer is quoted as causing 0.5 to 2 per cent of all deaths from cancer.

AGE

Again with the exception of India [*22*], there is general agreement in the literature that lingual cancer is predominantly a disease of the sixth and seventh decades of life [*2,4,6,11,17, 18,20,21,27*]. Our study supports these figures since 59 per cent of our patients were in their fifties and sixties. The youngest patient in the entire group was twenty-three years of age (present study) and the oldest was ninety-three [*4*]. Five per cent of the patients were less than forty years of age and twenty-three per cent were seventy or more. With the elimination of *in situ* and untreated lesions, 51 per cent of our thirty-seven traced patients less than fifty years of age were alive after five years as compared to only 38 per cent of the 151 traced patients fifty years of age or more (Table 1), which is contrary to the generalization that cancer tends to have a poorer prognosis in younger patients.

TABLE 1

EPITHELIOMA OF THE TONGUE: FIVE YEAR SURVIVAL RATES BY AGE AND SEX

Age (yr.)	Total				Men				Women			
	Patients		Lived 5 Years or More after Treatment		Patients		Lived 5 Years or More after Treatment		Patients		Lived 5 Years or More after Treatment	
	Total	Traced	Number	Per cent*	Total	Traced	Number	Per cent*	Total	Traced	Number	Per cent*
<50	37	37	19	51.4	31	31	15	48.4	6	6	4	66.7
50 or more.	157	151	57	37.7	116	111	37	33.3	41	40	20	50.0
Total....	194	188	76	40.4	147	142	52	36.6	47	46	24	52.2

* Based on traced patients; inquiry as of January 1, 1959.

SEX

The predominance of men over women having this disease has been reported to be 60 to 90 per cent [*1,4,8,11,13,14,17,18,20,22,23,27*]. In our study 75 per cent of the patients were men. The difference in *incidence* of lingual carcinoma in the two sexes is as noteworthy as is the *prognosis* in men as compared to that in women (Table 1). A total of 37 per cent of the 142 traced men with infiltrating epitheliomas were alive five years or more later, whereas 52 per cent of the forty-six traced women with infiltrating disease lived five years or more.

SIGNS AND SYMPTOMS

Pain, either in the tongue or elsewhere, is the commonest complaint in this disease; it was present in 147 (68 per cent) of our 217 patients and was the presenting symptom in 134 of them. Painless lesions of the tongue were noted by eighty patients, and twenty-five patients had a painless mass in the neck as all or part of the initial complaint. Loss of weight was not a predominant feature of this disease since only seventeen patients reported this finding. In only seven patients was the condition asymptomatic. Whereas pain usually is the first symptom, it is not in all probability an early complaint, since the survival rate of patients with pain as the initial symptom was less than the over-all survival rate in infiltrating carcinoma.

In only 8 per cent of the patients with pain was a positive diagnosis made within one month. On the other hand, the condition in 25 per cent of the patients with painless lesions and 16 per cent of those with a mass in the neck was diagnosed within a month. This finding leads one to question the widely accepted concept that pain is the prime factor in motivating a patient to seek medical attention.

One wonders what effect a delay in treatment from the onset of the first symptom has on survival. Of the seven patients in whom the disease was discovered incidentally, five survived for five years and only one of the two deaths was caused by this disease. A total of 49 per cent of those who began treatment less than two months after the onset of the initial symptoms survived for five years or more, whereas 30 per cent of those treated within two to six months survived for five years and only 20 per cent of the patients treated within six to eighteen months survived for five years. Thus, it appears that initiating treatment within two months of the onset of the initial symptoms can salvage an additional 20 to 30 per cent of these patients.

TREATMENT

To evaluate the efficacy of treatment for epithelioma of the tongue, especially with regard to the effect of palliation in prolonging life, one must know what the natural course of this disease would be if treatment were not instituted. Marcial [*18*] reported an average survival of three months after diagnosis of twenty untreated patients; Figi [*14*] stated

TABLE II
EPITHELIOMA OF THE TONGUE: FIVE YEAR SURVIVAL RATES ACCORDING TO PLACE OF TREATMENT

Place Treated	Patients		Lived 5 Years or More after Treatment	
	Total	Traced	Number	Per cent*
Mayo Clinic	153	149	63	42.3
Elsewhere	10	9	1	11.1
Both	31	30	12	40.0
Total	194	188	76	40.4

* Based on traced patients; inquiry as of January 1, 1959.

that death would ensue in approximately one year if the disease was allowed to progress untreated.

In our study ten patients did not receive treatment. One of these patients died from a bleeding gastric ulcer and one from cardiac arrest before treatment could be instituted. The remaining eight patients elected to return home without treatment. They apparently never received any form of therapy. One patient lived eighteen months, one survived for one year and the remaining six died within six months. The longest period that any of these patients had been symptomatic was two years and the shortest was eight months. Consequently, eighteen months would be a reasonable estimate for the duration of the natural course of this disease. The fact that many patients survived for three or four years after palliative treatment indicates the probable value of such therapy and, incidentally, the folly of using three year survival rates in the evaluation of this disease.

In Situ Lesions. Of the thirteen patients (6 per cent) who had *in situ* lesions, nine were treated at the clinic. None had evidence of cervical metastasis and all survived for five years or more. However, one patient treated elsewhere subsequently had infiltrating disease and died with cervical metastasis; this case serves as a reminder that, even in the presence of so-called minimal involvement, this lesion definitely has lethal potentialities.

Infiltrative Disease. A total of 153 patients with infiltrating lingual carcinoma received their entire treatment at the clinic. (Table II.) In the lesions in these patients, the ratio of anterior to basilar involvement was approximately 1:1. The degree of malignancy in 55 per cent of the lesions was graded 3 or 4, according to Broders' classification [7], and in only 18 per cent of cases was the primary tumor less than 2 cm. in diameter at the time of treatment. Local recurrence of the epithelioma took place in twenty-seven patients (18 per cent), and sixty-three of the traced patients (42 per cent) survived for five years or more. Results of treatment of the thirty traced patients who were treated only in part at the clinic (40 per cent surviving for five years or more) also were encouraging.

The survival rates according to treatment (Table III) indicate that electrocoagulation in some cases is an excellent method of attacking the primary lesion. This treatment is employed only in selected instances, however, as is indicated by the fact that 71 per cent of the lesions in this group were of a low grade of malignancy.

Combinations of treatment are occasionally helpful. Comparison of the survival figures in our group might lead to the conclusion that sharp dissection is the treatment of choice. However, before any true comparison of various available therapeutic methods can be made, factors other than treatment must be eliminated. Thus, whereas 44 per cent of the patients treated by sharp dissection survived for five years or more as compared with only 16 per cent of those managed by irradiation, less than half of the lesions in the group treated with surgery (46 per cent) were of a high grade of malignancy compared to an 82 per cent incidence of high grade lesions in the group treated by irradiation. Although 72 per cent of the tumors in the surgical group were more than 2 cm. in diameter, all of the lesions treated by irradiation were in this category. If, in comparing results of surgical treatment and irradiation of the primary disease, we eliminate factors other than treatment that change survival time and select truly comparable lesions (all of the same grade, all infiltrating, all more than 2 cm. in diameter and all with metastatically involved cervical lymph nodes), there is apparently little difference in prognosis regardless of whether surgical dissection or irradiation is chosen for treatment of the primary disease. (Table IV.)

In the management of cervical metastasis,

TABLE III

EPITHELIOMA OF THE TONGUE: FIVE YEAR SURVIVAL RATES BY TYPE OF TREATMENT

Type of Treatment	All Treatment at Mayo Clinic				Part of Treatment at Mayo Clinic			
	Patients		Lived 5 Years or More after Treatment		Patients		Lived 5 Years or More after Treatment	
	Total	Traced	Number	Per cent*	Total	Traced	Number	Per cent*
Primary treatment of local lesion								
Sharp dissection	52	50	22	44.0	14	14	9	64.3
Electrocoagulation	16	16	10	62.5	2	2	1	50.0
Irradiation	38	37	6	16.2	7	7	1	14.3
Sharp dissection and electrocoagulation	12	11	7	63.6	0	0	0	...
Sharp dissection and irradiation	24	24	13	54.2	4	4	1	25.0
Electrocoagulation and irradiation	11	11	5	45.4	4	3	0	...
Total	153	149	63	42.3	31	30	12	40.0
Primary treatment of neck lesion								
Sharp dissection	88	86	42	48.8	7	7	4	57.1
Irradiation	49	48	11	22.9	19	18	4	22.2
None	16	15	10	66.7	5	5	4	80.0
Total	153	149	63	42.3	31	30	12	40.0

* Based on traced patients; inquiry as of January 1, 1959.

TABLE IV

EPITHELIOMA OF THE TONGUE: FIVE YEAR SURVIVAL RATES FOR SURGICAL TREATMENT AND IRRADIATION

Treatment	Primary Lesion Grade 3, Infiltrating, More Than 2 cm., Involved Nodes				Neck Lesion Grade 3, Infiltrating, Involved Nodes			
	Patients		Lived 5 Years or More after Treatment		Patients		Lived 5 Years or More after Treatment	
	Total	Traced	Number	Per cent*	Total	Traced	Number	Per cent*
Sharp dissection	20	20	3	15.0	33	33	10	30.3
Irradiation	16	16	2	12.5	26	25	2	8.0
Total	36	36	5	13.8	59	58	12	20.7

* Based on traced patients; inquiry as of January 1, 1959.

TABLE V
EPITHELIOMA OF THE TONGUE: FIVE YEAR SURVIVAL RATES FOR EN BLOC REMOVAL AND SUPRAHYOID DISSECTION

Treatment	Patients		Lived 5 Years or More after Treatment	
	Total	Traced	Number	Per cent*
En bloc removal	18	17	9	52.9
Suprahyoid dissection	31	30	22	73.3
Total	49	47	31	65.9

* Based on traced patients; inquiry as of January 1, 1959.

however, the picture is quite different. In comparable lesions, surgical dissection clearly offers a far better chance of surviving five years than does irradiation. (Table IV.)

"Commando" Procedures. Since the *en bloc* dissection of epitheliomas involving the tongue and neck (the so-called commando or pull-through procedure) has lacked wide acceptance until relatively recent times, the literature reporting five year follow-up data for this operation is sparse [*23,28*]. Our series also is small, since this procedure has been used extensively at the clinic only in the past six or seven years. (Table V.) Of eighteen patients on whom this type of operation was performed, nine survived for five years or more; however, only two of these patients had proved cervical metastasis.

Suprahyoid Dissection. Suprahyoid dissection during the early course of this study was rather common; of a total of thirty-one such dissections, twenty-seven were bilateral and four were unilateral. (Table V.) Only one patient had involved lymph nodes found at operation; in this instance, the procedure was curative. Subsequently, nodal involvement developed in nine more patients and required further management. Of thirty traced patients having suprahyoid dissection, twenty-two survived for five years or more. We think that the indications for this type of surgical treatment are limited.

Prophylactic Dissection of the Neck. Prophylactic dissection of the neck was performed in fifty-two of the 119 patients (44 per cent) thought to be free of cervical metastasis on admission. It is evident that a certain amount of selectivity entered into the decision to proceed with this operation even though enlarged lymph nodes were not palpated, since this procedure was not undertaken in sixty-seven cases. Much of this selection undoubtedly was based on the size of the lesion and, perhaps to a lesser extent, on the condition of the patient. Of the fifty-two patients on whom this prophylactic operation was performed, nineteen (37 per cent) had microscopic evidence of nodal spread; six of these nineteen patients lived for five years or more. Metastatically involved nodes subsequently appeared in ten of the sixty-seven patients in whom the neck was not attacked surgically; six of these ten patients died of the disease. Had routine prophylactic dissection of the neck been performed initially on all 119 patients thought to be free of involved nodes, it is possible that we could have salvaged an additional two patients (1.7 per cent). This figure, however, closely approximates the operative mortality rate of 4 per cent for radical operations on the neck quoted in the literature.

SUMMARY AND CONCLUSIONS

A study has been made of 217 cases of squamous cell carcinoma of the tongue seen at the Mayo Clinic in the five year period from 1949 through 1953.

Squamous cell carcinoma of the tongue comprises approximately 0.8 per cent of all new cases of cancer and is responsible for 0.5 to 2 per cent of all deaths from cancer.

Fifty-nine per cent of these 217 patients were in the sixth and seventh decades of life. A total of 75 per cent of these patients were men. Women have a somewhat better prognosis.

Pain is the commonest complaint, but it is not an early symptom. A period of eighteen months appears to be a reasonable estimate for the duration of the natural course of the disease.

If treatment had been instituted in all cases within two months after the initial onset of symptoms, an additional 20 to 30 per cent of the patients perhaps could have been salvaged. Although all the patients with *in situ* disease treated at the clinic survived for five years or more, this form of carcinoma is still a disease with lethal potentialities.

The five year survival rate of the 149 traced patients with infiltrative disease treated en-

tirely at the clinic was 42 per cent; the rate among thirty traced patients treated in part here was 40 per cent.

In comparable primary disease (grade 3 infiltrating lesions more than 2 cm. in diameter with involved lymph nodes) little difference exists in the results of surgical dissection versus irradiation. When comparable disease has involved the neck, however, surgical dissection is clearly the treatment of choice.

Eighteen patients had *en bloc* resection, and nine of them survived for five years or more; however, only two of these patients had proved cervical metastasis.

Suprahyoid dissection rarely is indicated in the management of this disease. Prophylactic dissection of the neck is not recommended as a routine procedure but is of value in selected instances.

REFERENCES

1. Ackerman, L. V. and del Regato, J. A. Cancer: Diagnosis, Treatment and Prognosis. 2nd ed., pp. 254, 371. St. Louis, 1954. C. V. Mosby Co.
2. Ariel, I. M. Treatment of tumors of the tongue. In: Pack, G. T. and Ariel, I. M. Treatment of Cancer and Allied Diseases: Tumors of the Head and Neck, 2nd ed., vol. 3, p. 101, New York, 1959. Paul B. Hoeber, Inc.
3. Baud, J. End results of radiotherapy of cancer of the tongue. *Am. J. Roentgenol.*, 63: 701, 1950.
4. Berridge, J. E., Jr. and James, A. G. The management of lingual cancer. *Surg. Gynec. & Obst.*, 103: 595, 1956.
5. Berven, E. End results of treatment of cancer of the tongue. *Am. J. Roentgenol.*, 63: 712, 1950.
6. Breed, J. E. Radium treatment of cancer of the tongue. *Radiology*, 69: 214, 1957.
7. Broders, A. C. The microscopic grading of cancer: the variability of the malignancy of cancers. *S. Clin. North America*, 21: 947, 1941.
8. Brown, J. B. and Fryer, M. P. The mouth, tongue, jaws and salivary glands. In: Christopher's Textbook of Surgery, 7th ed., p. 270. Philadelphia, 1960. W. B. Saunders Co.
9. Cade, S. Cancer of mouth and tongue. *Northwest Med.*, 56: 1431, 1957.
10. Cade, S. and Lee, E. S. Cancer of the tongue: a study based on 653 patients. *Brit. J. Surg.*, 44: 433, 1957.
11. Catlin, D. Cancer of the tongue. *New York J. Med.*, 58: 2703, 1958.
12. Dorn, H. F. and Cutler, S. J. Morbidity from Cancer in the United States. (Monograph No. 56.) U. S. Department of Health, Education and Welfare, Public Health Service, 1959.
13. Farr, H. W. Cancer of the tongue. *Am. J. Nursing*, 57: 1314, 1957.
14. Figi, F. A. Malignant diseases of the mouth. *J. Am. Dent. A.*, 23: 216, 1936.
15. Gibbel, M. I., Cross, J. H. and Ariel, I. M. Cancer of the tongue: a review of 330 cases. *Cancer*, 2: 411, 1949.
16. Lyall, D. and Schetlin, C. F. Cancer of the tongue. *Ann. Surg.*, 135: 489, 1952.
17. Marchetta, F. C. and Mattick, W. L. Carcinoma of the tongue: treatment and results without radical surgery. *Surgery*, 40: 378, 1956.
18. Marcial, V. A. Carcinoma of the base of the tongue. *Am. J. Roentgenol.*, 81: 420, 1959.
19. Martin, C. L. Treatment of cancer of the tongue and its cervical metastases with irradiation. *South. M. J.*, 47: 1, 1954.
20. Palmer, J. D., Lougheed, M. N. and Martin, S. J. Carcinoma of the tongue: a study of 100 cases at the Montreal General Hospital. *Canad. J. Surg.*, 2: 180, 1959.
21. Passalacqua, L. A. Carcinoma of the base of the tongue. *Bol. Asoc. méd. Puerto Rico*, 49: 268, 1957.
22. Paymaster, J. C. and Shroff, P. D. The problem of carcinoma of the tongue in India. *Am. J. Surg.*, 94: 450, 1957.
23. Perzik, S. L., Joergenson, E. J., Carter, R. P. and Deeb, P. H. End-results in carcinoma of the oral cavity: a study based upon six hundred seventy-nine cases over a fifteen-year period. *Arch. Surg.*, 76: 677, 1958.
24. Shedd, D. P., Schmidt, N. L. and Chang, C. H. A survey of tongue cancer over a fifteen year period in a general hospital. *Surg. Gynec. & Obst.*, 106: 15, 1958.
25. Southwick, H. W., Otten, J. W. and Slaughter, D. P. Treatment of squamous cell carcinoma of the tongue. *Surgery*, 43: 313, 1958.
26. Texas Cancer Coordinating Council. The tongue and floor of the mouth. *Cancer Bull.*, 9: 94, 1957.
27. Trieger, N., Ship, I. I., Taylor, G. W. and Weisberger, D. Cirrhosis and other predisposing factors in carcinoma of the tongue. *Cancer*, 11: 357, 1958.
28. Wawro, N. W. and Babcock, A. Treatment of cancer of the tongue at Hartford Hospital. *New England J. Med.*, 258: 317, 1958.
29. Wilson, J. L. and Brizzolara, L. G. Carcinoma of the tongue. *Ann. Surg.*, 136: 964, 1952.
30. Windeyer, B. W. End results and treatment of cancer of the tongue. *Am. J. Roentgenol.*, 63: 719, 1950.
31. Wynder, E. L., Bross, I. J. and Feldman, R. M. A study of the etiological factors in cancer of the mouth. *Cancer*, 10: 1300, 1957.

Hirschsprung's Disease

A NEW MODIFICATION FOR RECTOSIGMOIDECTOMY

FAYEK GHABRIAL, M.B., B.CH., M.CH., *Cairo, United Arab Republic*

From the Department of Surgery, Kasr-el-Aini University Hospital, and Children University Hospital and the Department of Medicine, Cairo University, Cairo, United Arab Republic.

HIRSCHSPRUNG'S disease or congenital megacolon is a condition characterized by a congenital defect in the formation of the parasympathetic system supplying the distal colon. This defect is the absence of the ganglion cells in Auerbach's plexus of the affected part. The extent of the aganglionic segment varies in various cases, but it always involves the rectosigmoid and the rectum, and extends down to and even involves the internal sphincter. It may extend more proximally to involve portions of colon supplied by the parasympathetic fibers of the vagus; less commonly the whole colon may be thus affected and in rare instances the condition can extend to the small intestine [2].

DIAGNOSIS

It is easy to detect histologically this congenital absence of the ganglion cells in Auerbach's plexus of the aganglionic segment of the colon. The ganglion cells can be demonstrated in sections stained with hematoxylin and eosin. They are found in clusters in between the layers of circular and longitudinal muscles. They are large-sized with finely granular cytoplasm, large nucleus and prominent nucleolus. They can also be identified accurately enough in frozen sections, a fact which is made use of during operation to assure the complete removal of the aganglionic segment.

An infant with idiopathic congenital megacolon (Hirschsprung's disease) manifests a rather unmistakable clinical picture. Constipation occurs from the time of birth, and there may be difficulty and delay in the first passage of meconium. This condition persists and progresses. The abdomen becomes protuberant and distended with flaring of the costal margins. Elevation of the diaphragm may be so marked as to simulate eventration. Distension may be so marked as to cause respiratory embarrassment. There is always the need for enemas to evacuate the bowel, and rarely do feces pass spontaneously and then in the form of hard scybala. Flatus may, however, be passed in annoyingly large quantities. Pain and vomiting are not marked features. There is no incontinence of feces or soiling of babies' clothes. The tone of the sphincter is normal. The rectal ampulla is empty. Ulceration of the colon may occur as a result of stasis. This may lead to the occurrence of tarry diarrhea which may cause some clinicians, especially general practitioners, to shrink from diagnosing Hirschsprung's disease which is associated in the minds of most if not all physicians with obstinate constipation. A barium enema, if properly carried out, is helpful in the diagnosis. A small amount of barium is injected and observed under the screen with the patient in the oblique or lateral position. The flow of barium should be stopped as soon as the dilated colon is partially filled; otherwise, the completely filled dilated sigmoid might obscure the outline of the narrowed rectum and rectosigmoid, especially in the anteroposterior view. Typically, there is a definite narrowing of the rectum and rectosigmoid involving at least 8 to 10 cm. associated with definite dilatation of colon proximal to the narrow segment. The child is then allowed to evacuate; however, he typically is unable to void completely, a finding which should be recorded on a postevacuation film. Finally, rectal muscle biopsy [9] will confirm the diagnosis and is a safe procedure.

The history, the clinical picture, the barium enema studies and the rectal muscle biopsy will all help to differentiate idiopathic congenital megacolon, due to the presence of an aganglionic segment, from other conditions associated with dilated colon; e.g., pseudomegacolon and obstinate constipation that may be due to psychogenic causes, or may occur in mentally deficient children or congenital cretins or as a result of the presence of organic anal obstruction.

TREATMENT

As a result of Swenson's [*5–8,11*] extensive work on this subject, the treatment of congenital idiopathic megacolon has been firmly established on a scientifically sound basis. Apart from some cases in which a preliminary colostomy [*10*] may be indicated, Swenson's method of treatment is a pull-through excision of the aganglionic segment.

After a proper and thorough preoperative preparation, the patient is placed in the lithotomy position. An indwelling catheter is passed into the bladder. The abdomen is opened through a left paramedian incision. The peritoneum of the pelvic floor is freed all around the circumference of the bowel. The dissection is carried down to about three fourths of an inch from the pectinate line, keeping directly on the rectal wall all the time in order to preserve the nerves leading to the bladder and to the ejaculatory mechanism. The bowel is then divided at the lowest functional point on the colon. The two ends of the bowel are rapidly but securely closed. The blood supply to the proximal bowel must be elongated to reach the perineum without the slightest risk of devitalizing the gut. This is carried out by dividing one or two lower branches of the inferior mesenteric artery, at the same time preserving the arterial arcades. Then the aganglionic segment is everted through the dilated anus. An incision is subsequently made in the wall of the everted rectum about 1½ inches from the pectinate line. An instrument is passed up through this incision and the normal colon is brought back down with it. An anastomosis is then carried out between the wall of the rectum and the lower end of the normal bowel, and the incision in the rectal wall is gradually carried around the wall of the bowel followed closely by the anastomotic procedure. After securing complete hemostasis, the pelvic floor is reconstructed and the abdominal wall is closed.

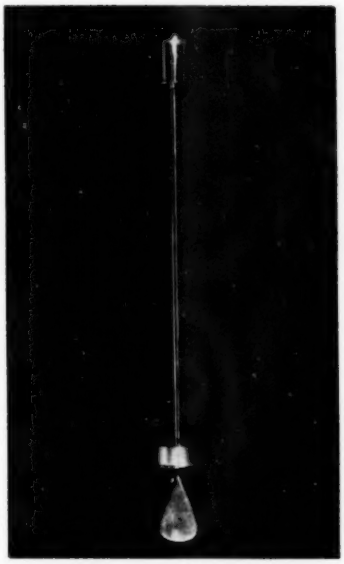


FIG. 1. Obturator used in the operation.

To avoid division of the bowel wall before pulling it through, a procedure which may possibly contaminate the pelvic cavity with subsequent postoperative complications of infection, some modifications have been suggested.

In Hiatt's [*4*] modification, an assistant inserts an Allis' clamp through the anus into the midrectum where a portion of the wall is grasped. With gentle traction the lower rectum is turned inside out. Repetition of this maneuver easily effects a complete eversion or intussusception of the entire rectum and lower two thirds of the sigmoid through the anus; but in spite of this modification, Hiatt reported complications of infection in seven of twenty-three patients operated on in this manner [*4*].

Brown [*3*] has modified this method: a sigmoidoscope is passed well up into the sigmoid loop by an assistant. Two special needles, 14 inches long, are then inserted through the end of the sigmoidoscope carrying with them two threads which are looped on the bowel. The result is that when the sigmoidscope and the needles are withdrawn, two traction threads are available to prolapse the sigmoid through the anus.

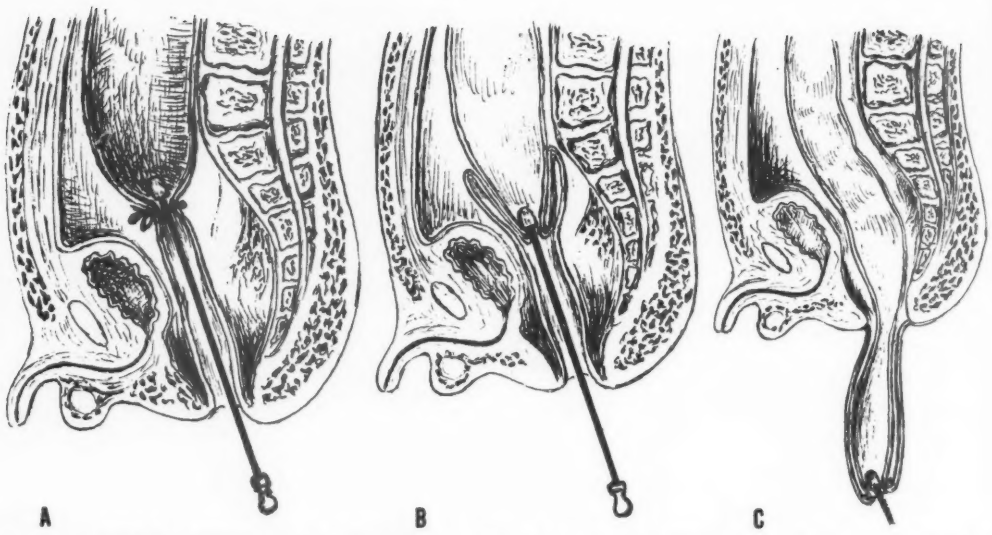


FIG. 2. A, thick silk ligature applied around the bowel on the stem of the obturator below its head. B, starting intussusception of the colon into rectum. C, intussusception completed.

In Behairy's technic [1], a wide thick-walled rubber rectal tube is passed through the anus, guiding it up to the level of the proximal resection. Then a thick silk thread on a needle is passed through the bowel wall and the rectal tube and tied. This stitch is repeated at four equidistant areas around the circumference of the bowel. When the rectal tube is pulled, the proximal colon is intussuscepted into the rectum and anal canal and everted.

It should be noted that in Hiatt's technic, there is traumatization by the sharp teeth of Allis' clamp which probably predisposes to postoperative complications of infection. It should also be noted that in both Brown's and Behairy's technics there is actual penetration into the lumen of the bowel by the needles. To intussuscept the freed colon through the rectum and anal canal and at the same time avoid these possible disadvantages, the following simple method is suggested.

A sigmoidoscopic obturator, of suitable size (Fig. 1), is passed a suitable distance up into the rectum, then a thick silk ligature is tied around the bowel on the stem of the obturator just below its head. (Fig. 2A.) Continuous and gentle traction on the obturator assisted by judicious manipulations from above results in easy intussusception of the colon into the rectum and anal canal. (Fig. 2A and B.) This method was first used in May 1959, and in three cases since. No difficulty was encountered during operation and no postoperative complications of infection followed. Although the number of cases is relatively small, it is believed that this modification is justifiably recommended.

SUMMARY*

1. A short account of the etiology, clinical picture and treatment of congenital idiopathic megacolon (Hirschsprung's disease) is given.

2. A new modification in the technic of rectosigmoidectomy for this disease is suggested.

REFERENCES

1. BEHAIRY, G. E. Congenital anomalies of colon, rectum and anus. *Kasr-el-Aini J. Surg.*, 1: 242, 1960.
2. BODIAN, M., CARTER, C. O. and WARD, B. C. H. Hirschsprung's disease, with radiologic observations. *Lancet*, 1: 302, 1951.
3. BROWN, D. Cited by Goligher, J. C. Rectosigmoidectomy for Hirschsprung's disease. In: Operative Surgery, vol. 3, part 4, p. 56. London, 1957. Butterworth & Co.
4. HIATT, R. B. The surgical treatment of congenital megacolon. *Ann. Surg.*, 133: 321, 1951.

* Since the time of submitting this article for publication, five more patients have been operated upon in this manner with the same results, namely, no difficulty during the operation and no postoperative infection.

5. SWENSON, O. A new surgical treatment for Hirschsprung's disease. *Surgery*, 28: 371, 1950.
6. SWENSON, O. Modern treatment of Hirschsprung's disease. *J. A. M. A.*, 154: 651, 1954.
7. SWENSON, O. and BILL, A. H., JR. Resection of rectum and recto-sigmoid with preservation of sphincter for benign spastic lesions. *Surgery*, 24: 212, 1948.
8. SWENSON, O. and FISHER, J. H. Resection of the colon for Hirschsprung's disease. *S. Clin. North America*, 36: 821, 1956.
9. SWENSON, O., FISHER, J. H. and MACMAHON, H. E. Rectal biopsy as an aid in diagnosis of Hirschsprung's disease. *New England J. Med.*, 253: 632, 1955.
10. SWENSON, O. and RHEINLANDER, H. F. Indications for colostomy in patients with Hirschsprung's disease. *Surg. Gynec. & Obst.*, 95: 738, 1952.
11. SWENSON, O., SEGNITZ, R. H. and SHEDD, R. H. Hirschsprung's disease. New surgical treatment. *Am. J. Surg.*, 81: 341, 1951.

LIBRARY
BOSTON UNIVERSITY
SCHOOL OF MEDICINE

Metastatic Carcinoma of the Mandible with Primary Tumor in the Lung

JACQUES E. R. VREBOS, M.D., JAMES K. MASSON, M.D. AND EDGAR G. HARRISON, JR., M.D.,
Rochester, Minnesota

From the Mayo Clinic and Mayo Foundation, Rochester, Minnesota.

FEW reports in the medical literature deal expressly with metastatic tumors of the jaws, which are to be sharply distinguished from involvement of the jaws by direct invasion from a contiguous malignant lesion originating in adjacent structures. The latter type of involvement occurs with relative frequency and seldom causes any diagnostic uncertainty. The following case of a metastatic tumor of the mandible with proved primary cancer of the lung is of interest because the condition is uncommon and it was not suspected early even though it was the initial presenting complaint.

CASE REPORT

On August 10, 1959 a fifty-six year old white man registered at the Mayo Clinic with a history of apparent good health until March 1959 when

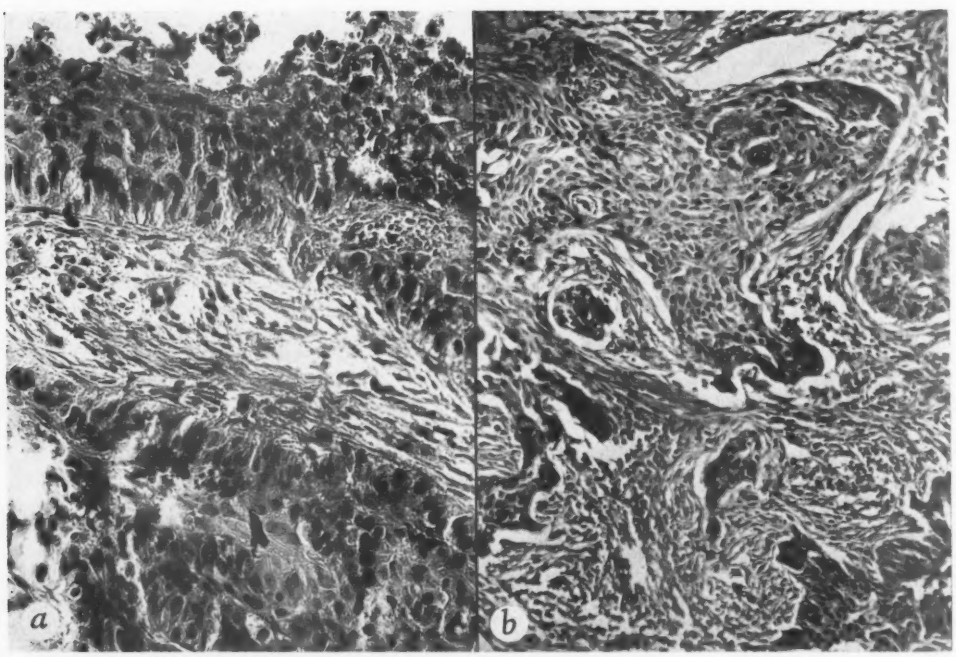


FIG. 1. A, biopsy of bronchial tissue showed squamous cell carcinoma, grade 3, lining the bronchus (upper) with infiltrating nests such as those shown in the lower portion of the photomicrogram (hematoxylin and eosin; × 250). B, tissue from left lung (pneumonectomy) showed infiltrative nests of squamous cell carcinoma, grade 3, and surrounding fibrosis of parenchyma of the lung. Note pearly body (upper) (hematoxylin and eosin; × 100).

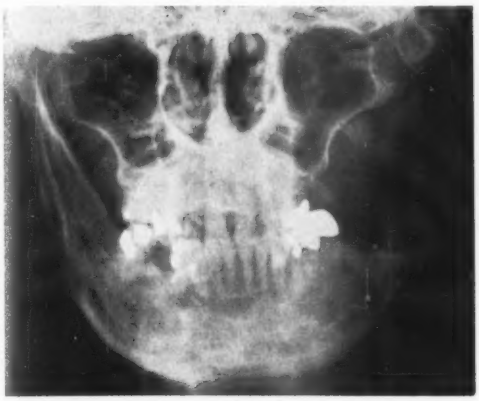


FIG. 2. Note the destructive lesion involving the horizontal ramus, angle, and ascending ramus of the left mandible.

he felt a dull ache in the left side of his face which felt at first like a pressure pain and later like a toothache. He contacted his dentist who ordered dental roentgenograms which revealed nothing of dental origin to account for the pain. Three weeks later, because of persistent discomfort in the left cheek, his home physician was consulted. He described a mass located in the left preauricular region and prescribed Achromycin®; but in spite of this the mass continued to enlarge and became hot and tender.

Concerned by the gradual enlargement of the mass and not satisfied with the treatment he was receiving, the patient sought another opinion. A diagnosis of parotitis was made and the patient was hospitalized. Sulfonamide drugs were administered, the parotid gland was massaged regularly and hot packs were applied. Despite continuous care the swelling increased and extended to involve the entire parotid and submandibular regions. On June 8, 1959 the patient suddenly coughed up bright red blood equal in amount to two tablespoonfuls. He was referred to the hospital and a roentgenogram of his thorax showed evidence of a tumor in the left lung. Bronchoscopy was performed and biopsy revealed a squamous cell epithelioma involving the left bronchus. (Fig. 1A.) On June 22, left pneumonectomy was performed and a squamous cell epithelioma was removed from the left bronchus. (Fig. 1B.) The tumor in the left cheek continued to increase in size and was assumed to be of an inflammatory nature. Treatment of the left side of the thorax with cobalt-60 was initiated on July 13; at the same time a series of three treatments of 450 r each were administered to the parotid regions to decrease the so-called inflammatory reaction of the parotid gland. The patient received his last treatment with cobalt-60 on

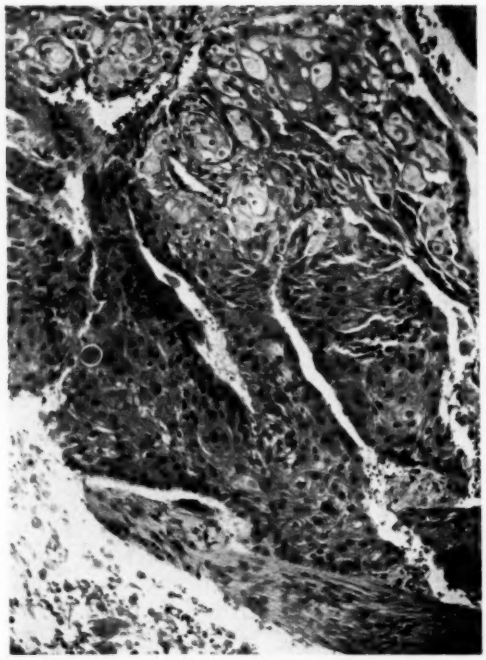


FIG. 3. Tissue from left mandible. Metastatic squamous cell carcinoma, grade 3, which was removed from deep within the mandible (hematoxylin and eosin; × 100).

August 4; immediately following this treatment he registered at the clinic for advice and for treatment of the swelling in the left cheek.

A large indurated mass involved the left side of the face and the mandible. The tumor measured approximately 9 cm. in diameter and was firm, slightly irregular, nontender and fixed to the mandible. It expanded the parotid region, the cheek, and the submandibular region and it extended into the neck at the angle of the mandible. The results of intraoral examination were essentially normal.

Roentgenograms of the mandible revealed an extensive malignant destruction of the ramus of the mandible on the left with an associated large, soft tissue mass; destruction extended down into the body of the mandible. There was no evidence of any bony involvement of the maxilla or the mandible on the left. (Fig. 2.)

At this stage, although the presence of a metastatic tumor was strongly suspicioned, only biopsy could give a positive answer. Biopsy was performed on August 15, 1959 under local anesthesia and the tumor was reported to be a metastatic squamous cell epithelioma, grade 3, compatible with the primary tumor in the lung. (Fig. 3.)

With the knowledge that bronchogenic carcinomas are inclined to cause skeletal metastasis

in 25 to 45 per cent of all cases, roentgenograms were made of the spinal column, skull and pelvis. None of them showed evidence of any abnormality.

A course of palliative x-ray therapy was outlined. The patient elected to receive the radiation therapy at a hospital near his home.

REVIEW OF LITERATURE

Because of the relative rarity of metastatic involvement of the jaws as recorded in the medical literature, we felt that a study of the reported case was important. About 220 cases of metastatic malignant lesions in both upper and lower jaws were found in the literature. It is highly possible that there are other instances of metastatic malignant lesions of the jaws which escaped our attention.

At the Mayo Clinic, Cash and Royer recently have analyzed and later will report the clinical data on metastatic tumors of the jaw bones from 1911 to 1958. They were able to trace twenty cases of proved metastatic tumor in the jaw bones. The sites of origin of reported cases of metastatic malignant lesions involving the jaws closely parallels the reported primary sites associated with generalized osseous metastasis. This is understandable since most of the reported instances of metastasis to the jaw have occurred in patients with generalized metastatic disease.

In the 220 cases reported in the literature the primary lesions most frequently accounting for metastasis to the jaw were as follows: carcinoma of the breast [*1–21*], fifty-one cases; carcinoma of the thyroid [*3,7,22–24*], forty-seven cases; hypernephroma [*18,25–37*], twenty-eight cases; carcinoma of the lungs [*3,15,18,19,36,38–44*], nineteen cases; carcinoma of the prostate [*3,13,18,20,32,45,46*], eleven cases; carcinoma of the lip [*13,32,45*], thirteen cases. Other primary lesions which had metastasized to the jaws included hepatoma [*47*], seminoma [*19*], melanoma [*32,45,48–50*], sarcoma of bone [*11,15,51–53*], neuroblastoma [*53*], lymphosarcoma [*19,29,54*], and Kaposi's sarcoma [*29*]. Metastatic lesions to the jaws also have been reported from carcinoma arising in the following organs: stomach [*3,29*], uterus [*18,55–57*], large intestine [*3,10,19,46,58–61*], parotid gland [*5*], tonsil [*13*], adrenal glands [*53,62,63*], testes [*64*], ovaries [*3,65–68*], and nasal mucous membranes [*45*].

The case we have reported is the only case of metastasis to the jaw secondary to lung cancer recorded at the Mayo Clinic. The entire medical literature contains reports of nineteen cases of lung cancers with metastasis in the upper or lower jaw. Dudits' account [*38*] of metastasis of lung cancer to the jaw in 1935 was the first to be recorded. The actual incidence of metastatic malignant lesions involving the jaws is probably considerably higher than reported, as most osseous metastatic lesions occur late in the course of the disease and complete x-ray examinations and necropsy, especially of the facial area, seldom are made.

It is a known fact, as emphasized by Ballantyne [*69*], that malignant cells of bronchogenic carcinomas frequently gain access to blood vessels. This ready access to the systemic circulation explains the frequency with which blood-borne metastasis complicates the course of bronchogenic carcinoma and explains the high incidence of widespread and sometimes bizarre distribution of the peripheral lesions [*70*]. Most authors agree that the reason the jaws are rarely affected is that they contain less red marrow and are less vascular than some of the other bones of the skeleton. In the age range of patients in which carcinoma prevails, there is usually a virtual absence of red bone marrow in the jaws [*71,72*].

We were able to review the reports of fifteen of the nineteen reported cases of metastasis of lung cancer to the jaw [*3,15,18,19,29,36,38–43*]. Although it was difficult to classify all lesions because of the unavailability of photomicrographs and lack of detailed information, we presumed that all were authentic and we considered them as such.

The most frequent jaw bone involved by such lesions is the mandible; the most frequent regions involved are the molar and premolar. Here the presence of cancellous bone is most abundant and this area also is in direct line with, and near the main branches of, the inferior dental artery [*16*]. Both of these features may have some bearing on the molar region as the most likely site for metastatic lesions of the jaws. The left side was involved in eight cases and the right side in seven cases. Three cases presented in females and twelve in males. The predominance of males is in relation to the greater frequency of cancer of the lungs in males.

SYMPTOMS

Although osseous metastasis generally occurs late in the known course of the disease, the

metastasis from lung cancer involving the jaw was the first symptom in ten of the fifteen cases reviewed. Metastasis to the jaw was manifested clinically through the development of (1) a tumor which may be a soft tumor of the gingiva as occurred in two cases, a mass in the jaw as occurred in four cases, or a growth from a tooth socket as occurred in four cases; (2) pain as was noted in seven cases (in three cases the tumor was painless); (3) symptoms referable to the teeth, such as toothache and loosening of the teeth; (4) pathologic fracture of the mandible (not reported but conceivable); or (5) neurologic symptoms due to involvement of the inferior alveolar nerve resulting in paresthesia, numbness or sensation of thickening of the lower lip.

Some tumors give rise only to a vague awareness of an indefinite complaint in the jaws or are entirely asymptomatic and the lesion is overlooked or may be found during a routine roentgenologic survey of bone if, for example, other osseous metastatic lesions have been discovered.

Other tumors of the jaw which might be found are primary oral epithelial or soft tissue tumors with osseous invasion, primary malignant lesions of jaw bone, benign tumors or cysts of the jaw, infection of dental origin, osteomyelitis and fibrous dysplasia.

ROENTGENOLOGIC APPEARANCE

In most cases metastatic carcinoma of the jaw produces evidence of bony lesions but the findings may be variable. Most lesions secondary to tumors of the lung are osteoclastic in nature; whereas, the typical metastatic lesion in the mandible is a unilocular, radiolucent, centrally located and fairly well circumscribed lesion. Roentgenograms with diffuse shadows suggesting necrotic bone and large areas of bone rarefaction with perforation of the bone cortex are encountered occasionally. The case presented herein was the only one in the series of metastatic lesions to the jaw from primary lung cancer which showed such extensive destruction of the entire vertical ramus of the mandible. In the maxilla the x-ray findings are difficult to define. In most of the reported cases destruction of the periodontal structures was present or no lesion was demonstrable.

Biopsy alone, if adequate and properly interpreted, is the only accurate method of establishing the diagnosis. Of the fifteen cases reviewed, two lesions were diagnosed as adenocarcinoma, three as anaplastic squamous cell epithelioma, and four as undifferentiated carcinoma; two lesions showed pleomorphic anaplastic malignant cells; one lesion showed infiltration of the epithelium by cancer cells. In three cases [3,38] no pathologic diagnosis was reported.

Treatment in all cases of metastatic malignant lesions of the jaws is palliative only. Moderate doses of conventional x-ray therapy may be used with the idea that the dosage should not be pushed to the point that it induces radiation necrosis or enhances the likelihood of pathologic fracture. Rarely, because of pain and a large fungating tumor, surgical intervention may be resorted to as palliative management.

Death usually ensues in a rather short time after the diagnosis of metastatic malignant lesion of the jaw, the time ranging from one month to one and one-half years in the cases reviewed. Most frequently death was due to other metastatic phenomena.

SUMMARY AND CONCLUSIONS

The case presented is the only case of metastatic tumor of the jaw secondary to lung cancer encountered at the Mayo Clinic. It is of interest because a well developed lesion in the mandible was first discovered as a dental complaint. The metastatic tumor of the jaw was present more than two months before any other clinical symptom of the primary disease and six months before the diagnosis of metastatic malignant lesion of the jaw. The patient was under medical care as soon as the dental complaints arose. This case is reported to draw attention to the fact that all types of bone tumors occur in the jaw. Metastatic malignant lesions involving the jaws should be emphasized as a possible development whenever a tumor is encountered in the jaw. They should be suspected in the differential diagnosis of tumors of the jaws whenever atypical sites or symptoms are present. Lichtenstein's statement that metastatic carcinoma is the commonest bone tumor when all tumors involving bone are considered can be denied or affirmed, as pertains to the jaws, only by conducting further investigation in which the jaws are more accurately considered. Diagnosis is usually made by exclusion

of a primary lesion in the jaw and confirmed by careful search for a primary focus and substantiated by histologic examination.

REFERENCES

1. Bagnoli, S. La malignità secondaria della ossa mascellari. *Arch. "de Vecchi" anat. pat.*, 20: 1003, 1954.
2. Adair, F. E. and Herrmann, J. B. Unusual metastatic manifestations of breast carcinoma. 1. Metastasis of breast carcinoma to mandible with report of five cases. *Surg. Gynec. & Obst.*, 83: 289, 1946.
3. Berg, A. Metastatische Kiefertumoren. *Wien. klin. Wchnschr.*, 49: 970, 1936.
4. Bigelow, N. H. and Walsh, T. S. Metastatic carcinoma of the mandible. *Ann. Surg.*, 137: 138, 1953.
5. Burford, W. N., Ackerman, L. U. and Robinson, H. B. G. Symposium on twenty cases of benign and malignant lesions of the oral cavity, from the Ellis Fischel State Cancer Hospital, Columbia, Missouri. *Am. J. Orthodontics*, 30: 353, 1944.
6. Burket, L. W. Case No. 54: jaw metastasis in primary mammary carcinoma. *Am. J. Orthodontics*, 27: 652, 1941.
7. Byars, L. T. and Sarnat, B. G. Mandibular tumors: a clinical, roentgenographic, and histopathologic study. *Surg. Gynec. & Obst.*, 83: 355, 1946.
8. Cadenat, F. M. and Parant, M. Métastase maxillaire d'un épithélioma du sein. *Rev. stomatol.*, 51: 578, 1950.
9. Carlier, G. Métastase maxillaire d'un cancer du sein. *Rev. stomatol.*, 49: 43, 1948.
10. Risak, E. Zur Klinik und Statistik der bösartigen Kiefergeschwülste. *Arch. klin. Chir.*, 147: 162, 1927.
11. Robinson, H. B. G. Metastasis of chondromyxosarcoma to the jaw and tooth. *Am. J. Orthodontics*, 33: 558, 1947.
12. Skillen, W. G. A case of metastatic carcinoma of the jaws. *J. Am. Dent. A.*, 17: 1678, 1930.
13. Thoma, K. H., Holland, D. J., Jr. and Rounds, C. E. Clinical Pathology of the Jaws: with a Histologic and Roentgen Study of Practical Cases. Springfield, 1934. Charles C Thomas.
14. Rogers, H. and Flo, S. Sarcoma of the breast: a report of twenty-two cases. *New England J. Med.*, 226: 841, 1942.
15. Aisenberg, M. S. and Inman, C. L. Tumors that have metastasized to the jaws. *Oral Surg.*, 9: 1210, 1956.
16. Blackwood, H. J. J. Metastatic carcinoma of the mandibular condyle. *Oral Surg.*, 9: 1318, 1956.
17. Moorman, W. C. and Shafer, W. G. Metastatic carcinoma of the mandible. *J. Oral Surg.*, 12: 205, 1954.
18. Salman, I. and Langel, I. Metastatic tumors of the oral cavity. *Oral Surg.*, 7: 1141, 1954.
19. Castigliano, S. G. and Rominger, C. J. Metastatic malignancy of the jaws. *Am. J. Surg.*, 87: 496, 1954.
20. Kronfeld, R. and Weinmann, J. P. Metastatic adenocarcinoma in the lower jawbone. *Arch. Path.*, 29: 683, 1940.
21. Stockdale, C. R. Metastatic carcinoma of the jaws secondary to primary carcinoma of the breast. *Oral Surg.*, 12: 1095, 1959.
22. Buxton, J. D. Carcinoma of the thyroid with metastases. *Proc. Roy. Soc. Med.*, 23: 946, 1940.
23. Ibrahim, H. and Schewket, F. Quoted by Castigliano, S. G. and Rominger, C. J. [19].
24. Ehrhardt, O. Zur Anatomie und Klinik der Struma maligna. *Beitr. klin. Chir.*, 35: 343, 1902.
25. Albrecht, P. Beiträge zur Klinik und pathologischen Anatomie der malignen Hypernephrome. *Arch. klin. Chir.*, 77: 1073, 1905.
26. Branch, C. G. and Norton, R. H. Metastatic hypernephroma of the jaw. *New England J. Med.*, 198: 559, 1928.
27. Clairmont, P. Ueber ein Hypernephrom-Impfrecidiv in den Bronchiallymphdrüsen. *Arch. klin. Chir.*, 73: 620, 1909.
28. Dechaume, M. Épithélioma métastatique du maxillaire inférieur d'origine dentaire. *Rev. stomatol.*, 34: 582, 1932.
29. Salman, I. and Darlington, C. G. Rare (unusual) malignant tumors of the jaws. *Am. J. Orthodontics*, 30: 725, 1944.
30. Holland, D. J. Metastatic carcinoma to the mandible. *Oral Surg.*, 6: 567, 1953.
31. Stein, G. Hypernephrommetastase als Epulis. *Deutsche Ztschr. f. Chir.*, 219: 318, 1929.
32. Lindeman, M. A. Diagnosis and treatment of benign and malignant tumors of the jaws and face. *J. Oral Surg.*, 11: 268, 1953.
33. Desaive, P. Les tumeurs "lentes" des maxillaires. *Acta chir. belg.* (supp.), 1: 27, 1955.
34. Kozoll, D. D. and Kirshbaum, J. D. Relationship of benign and malignant hypernephromoid tumors of kidney: clinical and pathological study of 77 cases in 12,885 necropsies. *J. Urol.*, 44: 435, 1940.
35. Sherman, R. S. and Pearson, T. A. The roentgenographic appearance of renal cancer metastasis in bone. *Cancer*, 1: 276, 1948.
36. Stern, L. and Shepard, A. Metastatic carcinoma of mandible. *J. Am. Dent. A.*, 28: 707, 1941.
37. Stewart, E. G. and Bruce, K. W. Mandibular tumor metastasized from a hypernephromea report of case. *J. Oral Surg.*, 11: 252, 1953.
38. Dudits, A. Quoted by Berg [3].
39. Jurgens, P. E. Metastatic carcinoma in the mandible. *Oral Surg.*, 7: 587, 1954.
40. Kwapis, B. W. and Kuebel, J. O. Bronchogenic carcinoma with probable metastasis to the gingiva: report of a case. *J. Oral Surg.*, 10: 255, 1952.
41. Seldin, H. M., Seldin, S. D. and Rakower, W. Metastatic carcinoma of the mandible: report of cases. *J. Oral Surg.*, 11: 336, 1953.
42. Stern, L. and Gottlieb, M. Metastatic tumor of the maxilla derived from carcinoma of lung. *J. Mount Sinai Hosp.*, 4: 452, 1938.
43. Waldron, C. W. Metastatic carcinoma of the mandible: adenocarcinoma of the lung with metastatic lesion of the mandible as first clinical sign. *Oral Surg.*, 5: 185, 1952.
44. Sterling, J. A., Tassman, G. C. and Goldsmith,

R. Solitary metastatic lesion to the gingivae masquerading as inflammation. *Oral Surg.*, 7: 403, 1954.

45. Geschickter, C. F. and Copeland, M. M. Tumors of Bone, 3rd ed., p. 472. Philadelphia, 1949. J. B. Lippincott Co.
46. Meyer, I. and Shklar, G. Involvement of the mandible and oral mucosa in a case of rectal adenocarcinoma with generalized metastases: report of a case. *Oral Surg.*, 11: 69, 1958.
47. Dick, A., Mead, S. G., Mensh, M. and Schatten, W. E. Primary hepatoma with metastasis to the mandible. *Am. J. Surg.*, 94: 846, 1957.
48. Bluestone, L. F. Malignant melanoma metastatic to mandible: report of a case. *Oral Surg.*, 6: 237, 1953.
49. DeCholnoky, T. Malignant melanoma: a clinical study of one hundred seventeen cases. *Ann. Surg.*, 113: 392, 1941.
50. Wilner, D. and Breckenridge, R. L. Bone metastasis in malignant melanoma. *Am. J. Roentgenol.*, 62: 388, 1949.
51. Goldstein, I. H. and Goldstein, M. C. Case no. 75: jaw metastases in chondroblastic osteogenic sarcoma. *Am. J. Orthodontics*, 29: 57, 1943.
52. Kemper, J. W. and Bloom, H. J. Metastatic osteochondroma of maxilla from primary tumor of tibia: report of case. *Am. J. Orthodontics*, 30: 704–708, 1944.
53. Burch, R. J. Metastasis of neuroblastoma to the mandible: report of case. *J. Oral Surg.*, 10: 160, 1952.
54. Pratt, V. H. and Pratt, C. I. A lymphosarcoma with metastasis. *J. Oral Surg.*, 9: 19, 1951.
55. Battaille, R. Le cancer secondaire du maxillaire inférieur. *Rev. stomatol.*, 52: 158, 1951.
56. Catania, A. F. Three nonvital teeth associated with chorioepithelioma: report of case. *J. Oral Surg.*, 11: 324, 1953.
57. Englert, R. J. and Pasqual, H. N. Metastatic chorioepithelioma of the gingival tissue: Report of a case. *Oral Surg.*, 10: 813–818, 1957.
58. Ivy, R. H. and Curtis, L. Metastasis of tumor of the rectum to the mandible. *Ann. Dent.*, 3: 133, 1936.
59. Bruce, K. W. and McDonald, J. R. Metastatic adenocarcinoma of the mandible from the sigmoid colon: report of case. *Oral Surg.*, 7: 772, 1954.
60. Hägglund, G. Adenocarcinoma of the mandible, a metastasis from a primary tumor in the sigmoid colon: report of a case. *Oral Surg.*, 12: 1228, 1959.
61. Bosco, H. F. Metastatic adenocarcinoma of the mandible. *Oral Surg.*, 11: 86, 1958.
62. Roman, B. Quoted by Castigliano, S. G. and Rominger, C. J. [19].
63. Young, G. Growth of right maxilla, (?) neuroblastoma, metastasis from suprarenals. *J. Laryng. & Otol.*, 50: 860, 1935.
64. Cameron, J. R. and Stetzer, J. J., Jr. Metastatic carcinoma of the mandible: report of case. *J. Oral Surg.*, 5: 227, 1947.
65. Van Huysen, G. and Wallace, W. R. J. Tumor metastasis in jaw, leading to recognition of primary tumor of ovary. *Dental Cosmos*, 74: 53, 1932.
66. Brown, J. B. and O'Keefe, C. D. Sarcoma of the ovary with unusual oral metastasis. *Ann. Surg.*, 87: 467, 1928.
67. Kostrubala, J. G., Thurston, E. W. and Chapin, M. E. Metastatic dysgerminoma of the mandible. *Oral Surg.*, 3: 1184, 1950.
68. Mueller, C. W., Topkins, P. and Lapp, W. A. Dysgerminoma of the ovary. *Am. J. Obst. & Gynec.*, 60: 153, 1950.
69. Ballantyne, A. J. Incidence of venous involvement in cancer of the lung. Thesis, Graduate School, University of Minnesota, 1956.
70. Abrams, H. L., Spiro, R. and Goldstein, N. Metastasis in carcinoma: analysis of 1,000 autopsied cases. *Cancer*, 3: 74, 1950.
71. Piney, A. Cancer of the bone-marrow. *Brit. J. Surg.*, 10: 235, 1922.
72. Cohen, B. Secondary tumours of the mandible. *Ann. Roy. Coll. Surgeons, England*, 23: 118, 1958.

Traumatic Rupture of the Normal Spleen

ANALYSIS OF EIGHTY-THREE CASES

TIMOTHY DENNEHY, M.D., *Somerville, Massachusetts,* TIMOTHY A. LAMPHIER, M.D., *Boston, Massachusetts,* WILLIAM WICKMAN, M.D., *Miami Beach, Florida,** AND RONALD GOLDBERG, B.S., *Marblehead, Massachusetts*

From the Boston City Hospital, Boston, Massachusetts, the St. Alexis Hospital, Cleveland, Ohio, and the Central Hospital, Somerville, Massachusetts.

THE STATISTICAL data from eighty-three cases of splenectomy for traumatic rupture of the spleen are presented in this paper. Although the literature is well endowed with studies of a similar nature, this series may be unique in that it consists of a large number of patients who, without any previous pathologic conditions of the spleen, sustained an accidental injury which caused a pure splenic rupture. It is our intention through careful examination of the data to set forth the clinical nature of a pure splenic rupture.

The cases in this study were derived from three urban hospitals and represent the admissions of sixty cases to the Boston City Hospital from 1938 to 1952, twenty-one cases to the St. Alexis Hospital, Cleveland, Ohio, from 1947 to 1952, and two cases to the Central Hospital, Somerville, Massachusetts, in 1956. Of these cases, 91 per cent were admitted between 1946 and 1956. (Table I.)

SEX AND AGE

The classical concept that the male sex is more prone to splenic injury than the female sex has been borne out by this series [*1,8,10,12*]. Over 70 per cent of the patients studied were of the male sex. Many explanations for this phenomenon have been offered such as the protective garments worn by the female sex to the more active life of the male sex. Yet, the true reason remains obscure.

It has been reported by many authors that the peak age incident of traumatic splenic rupture is the third through fifth decades of life. We have found that over 50 per cent of our cases were in the first two decades, while only 36 per cent were in the third, fourth and fifth decades [*8,13*].

Of those forty-four cases in the first two decades, 57 per cent (or 30 per cent of the total series) of the patients were one hour to nine years old. We have, therefore, found that the peak incidence of such splenic injury is in early childhood prior to ten years of age. (Table II.) It is also evident from the data that there is an indirectly proportional relationship between age and incidence which holds true for every decade of age to the age of eighty years.

The mortality rate in those patients between sixty and eighty years of age was found to be six times that found in any other age group encompassing twenty years. Therefore, although the incidence of splenic rupture is the least for persons over sixty years of age, the mortality rate in this age group is the highest. (Table III.)

In this study of eighty-three patients there were eleven deaths, one of which was a newborn baby who died one hour after birth. The over-

TABLE I
DATES OF ADMISSION

Dates	No. of Patients	Per cent of Series
1938 to 1941	4	5
1942 to 1945	4	5
1946 to 1949	23	28
1950 to 1953	29	35
1954 to 1956	23	28

* Present address: Mt. Sinai Hospital, Miami Beach, Florida.

TABLE II
SEX AND AGE INCIDENCE

Age (years)	No. of Patients			Per cent of Series
	Male	Female	Total	
0 to 9	18	7	25	30
10 to 19	15	4	19	23
20 to 29	9	7	16	19
30 to 39	5	3	8	10
40 to 49	6	0	6	7
50 to 59	3	1	4	5
60 to 69	2	1	3	4
Over 70	2	0	2	2
Total.....	60	23	83	100

all mortality rate in this series was 13 per cent and is compared with that of other series in Table IV.

CAUSES OF INJURY

Other studies have shown that splenic ruptures are more often caused by the blunt types of injuries than by the penetrating types [3,7,8]. We have found that 94 per cent of cases in our series were caused by a blunt injury. While this group had a mortality rate of 11 per cent, the group with a penetrating etiology had a 40 per cent mortality rate, indicating that while the penetrating wounds are the least common, they

TABLE IV
MORTALITY RATE OF SPLENIC RUPTURE

Author	No. of Cases	Mortality Rate (%)
Bessel-Hagen [1].....	27	46
Michelson.....	298	33
Buxton [5].....	...	29
McTudoc.....	37	27
Foster and Prey [6].....	20	30
Anderson.....	9	33
Wright and Prigot [18].....	30	43
Rousselot and Illyne [15].....	17	29
Zabinski and Harkins [19].....	10	30
Roettig et al. [14].....	15	7
Larghero and Guiria [9].....	18	6
Bollinger and Fowler [3].....	24	17
Parsons and Thompson [12]....	32	12
Knopp and Harkins [8].....	28	36
Mansfield [10].....	16	0
Present series.....	83	13

TABLE III
MORTALITY BY AGE GROUPS

Age (years)	No. of Patients	No. of Deaths	Mortality (%)
0 to 9	25	3	12
10 to 19	19	1	5
20 to 29	16	1	6
30 to 39	8	2	25
40 to 49	6	1	17
50 to 59	4	0	0
60 to 69	3	1	33
Over 70	2	2	100
Total.....	83	11	13

cause the greatest mortality. It is interesting to note that both of the patients, in whom injury was sustained by a bullet wound, died while the three patients injured by a stab wound survived.

Of the types of blunt injuries, the automobile is indicted for being the most common cause of splenic injury and for having the greatest mortality rate; 38 per cent of the patients in our series were so injured and 17 per cent of these died. While injuries from falling were almost as frequent as the automobile accidents (30 per cent of the cases), they claimed only half the mortality rate of the latter. Next in frequency were injuries due to blows and sledding, but all seventeen patients survived. (Table v.)

TABLE V
MODE OF INJURY AND MORTALITY

Mode of Injury	No. of Patients	Per cent of Patients with Known Etiology	No. of Deaths	Mortality (%)
Penetrating				
Stab.....	3	4	0	0
Bullet.....	2	3	2	100
Total.....	5	7	2	40
Blunt				
Automobile.....	30	38	5	17
Fall.....	24	30	2	8
Blow.....	9	11	0	0
Sledding.....	8	10	0	0
Cycling.....	2	3	0	0
Delivery.....	1	1	1	100
Total.....	74	93	9	11

A definite mode of injury was not recorded in only four of the eighty-three cases or 5 per cent. Of of these was a fifty year old man in whom a laceration developed in the splenic capsule over the dorsal diaphragmatic surface which was associated with acute pancreatitis. Numerous adherent blood clots had completely separated the spleen from the diaphragm. Following splenectomy, the patient was well.

Another was a twenty-one year old woman in whom a bleeding point near the hilus of the spleen in the direction of the angle developed, along with a ruptured ectopic pregnancy. She too was well following splenectomy.

In a fifty-six year old woman, a complete transection of the spleen into two pieces which hung loose from a small vascular pedicle developed. There were no associated illnesses present in this patient and in this as in the other two cases no history of injury could be elicited. This patient was well following splenectomy. No information as to etiology or pathology was available for the fourth case.

We are inclined to agree with others that these "spontaneous" cases are, in reality, the results of either a trivial unrecognized injury or one that had occurred some time previous to the rupture, an interval of time so long that the connection between the injury and the present illness (or the actual injury itself) was forgotten. This incidence would then fall into the category of latent splenic rupture.

ASSOCIATED INJURIES

That the incidence of associated injuries in patients with splenic rupture is high has been borne out by our study. In over three fourths of our patients there were injuries to other organs of the body. One third of the patients sustained injuries to the chest and head, and one seventh had fractures of the extremities or injuries to the kidneys. Injuries to the pancreas, liver, heart and gastrointestinal tract were rather rare. (Table VI.)

Since concussions, contusions and lacerations of the head as well as fractures of the extremities are quite common in most types of accidents in which the subject is in motion, and since these areas are not directly related to the splenic area, these injuries are not too useful in the clinical diagnosis of splenic rupture. Any injury to the chest, however, especially accompanied by evidences of damage to the kidney as manifested usually by macroscopic or microscopic hematuria should immediately cause the clinician to suspect a concomitant splenic injury.

TABLE VI
INCIDENCE OF ASSOCIATED INJURIES

Anatomic Parts Injured	No. of Patients	Total Series (%)
Chest	27	33
Head	25	30
Bones (fractures, excluding skull and ribs)	12	14
Kidney	11	13
Pancreas	5	6
Liver	5	6
Small bowel	5	6
Stomach	4	5
Heart	2	2
Colon	2	2
Omentum	2	2
Diaphragm	1	1
None	20	24

SYMPTOMS

Pain in the abdomen is the chief index of a splenic rupture. We have found that 40 per cent of those patients with symptoms had generalized pain in the abdomen when admitted. Pain localized in the left upper quadrant was present in 36 per cent and in the left lower quadrant in 26 per cent. Only about 10 per cent had symptoms pertaining to the right abdomen and epigastrium, while 14 per cent had pain in one or both shoulders. Symptoms pertaining to other areas of the abdomen represented but a few scattered cases. Nausea and vomiting are common in patients with this condition. Over one third of the total series had vomited at least once prior to surgery. (Table VII.)

It is interesting to note that of the eight patients who had no abdominal complaints at the time of admission, four died (a mortality rate of 50 per cent). Although it is possible for a rupture of the spleen to occur without symptoms, general or localized pain in the left side of the abdomen together with nausea and vomiting are so common that they might be considered the "splenic rupture syndrome."

SIGNS

Generalized abdominal tenderness and spasm together with hypoperistalsis are the predomi-

TABLE VII
SYMPTOMS

Symptoms	No. of Patients	Per cent of Patients
General abdominal pain	31	44
Localized abdominal pain		
Left upper quadrant	25	36
Left lower quadrant	18	26
Right upper quadrant	8	11
Epigastrium	6	9
Right lower quadrant	6	9
Paraumbilical	2	3
Suprapubic	1	1
Abdominal pain radiating to left or both shoulders	10	14
Nausea	26	31
Vomiting	30	36
None	8	11

TABLE VIII
SIGNS

Sign	No. of Patients	Per cent of Patients
Tenderness		
Generalized	31	40
Left upper quadrant	29	38
Rebound	20	26
Left lower quadrant	11	14
Right upper quadrant	8	10
Epigastrium	5	6
Right lower quadrant	2	3
Paraumbilical	1	1
Inguinal	1	1
Spasm		
General	35	45
Left upper quadrant	17	22
Left lower quadrant	5	6
Right upper quadrant	4	5
Left upper quadrant	1	1
Distension	10	13
Hypoperistalsis	26	34
Shifting dullness	5	6
Balance's sign	3	4
Kehr's sign	7	9
Fever	59	72
None	1	1
Not known	5	6

nant signs of this condition. About one half of those patients with demonstrable signs showed generalized tenderness or spasm and over one-third showed hypoperistalsis. The left upper quadrant revealed the typical signs of peritonitis due to internal hemorrhage. This was demonstrated as spasm or tenderness localized in this region in over one third of the patients. Pain in the shoulder (Kehr's sign) and dullness in the left upper quadrant (Balance's sign) were positive in only 4 and 9 per cent, respectively. (Table VIII.)

In 72 per cent of the patients a temperature of over 98.9°F. was present at the time of admission. Over two-thirds ranged from 99°F. to 100.9°F. (Table IX.) On admission, the systolic blood pressure in thirty-two patients was less than 100 mm. Hg. These patients constituted 45 per cent of those who had their blood pressure taken. (Table X.) Of these patients with evidences of shock, twelve were classified clinically as being in a state of severe vascular collapse. It was evident that as the shock increased in severity, the mortality rate rose. This was also reflected in the transfusion requirements, for as the requirements exceeded 2 L. of fluid, the mortality rate increased sharply. A definite correlation between the degree of shock and the mortality rate is, therefore, evident.

TABLE IX
TEMPERATURE ON ADMISSION

Temperature (°F.)	No. of Patients	Per cent of Patients with Known Temperatures
96.0 to 96.9	1	1
97.0 to 97.9	4	5
98.0 to 98.9	14	18
99.0 to 99.9	25	32
100.0 to 100.9	16	20
101.0 to 101.9	8	13
102.0 to 102.9	4	5
103.0 to 103.9	5	6
104.0	1	1
Not known	5	...

TABLE X
BLOOD PRESSURE (SYSTOLIC) ON ADMISSION

Blood Pressure (mm. Hg)	No. of Patients	Per cent of Patients with Known Pressure
Over 100	39	55
51 to 100	31	44
Under 50	1	1
Not known	12	...

TABLE XI
WHITE BLOOD CELL COUNTS AND MORTALITY

White Blood Cell Counts (10^3 per cu. mm.)	No. of Patients	Per cent of Patients with Known White Blood Cell Counts	No. of Deaths	Mortality (%)
6 to 10	14	17	2	14
11 to 15	24	30	2	8
16 to 20	13	16	1	8
21 to 25	17	20	2	12
Over 25	13	16	3	23
Not known	2	...	...	...

TABLE XII
RED BLOOD CELL COUNTS

Red Blood Cell Counts (10^6 per cu. mm.)	No. of Patients	Per cent of Patients with Known Red Blood Cell Counts
1.1 to 2.0	1	1
2.1 to 3.0	29	36
3.1 to 4.0	26	33
4.1 to 5.0	23	29
Over 5.0	1	1
Not known	3	...

LABORATORY STUDIES

Hematologic studies for the diagnosis of this pathology have been greatly stressed [*2,8,19*]. If the traditional upper limits of normality in white blood cell counts is taken as 10,000 per cu. mm., then of the eighty-one patients with known counts, 86 per cent had elevated white blood cell counts. Two thirds ranged between 11,000 and 25,000 per cu. mm. The mortality rate for those patients with counts of over 25,000 per cu. mm. was 23 per cent, while the average mortality rate for the other groups was about 10 per cent. (Table XI.)

Severe anemia (red blood cell counts of less than 3×10^6 per cu. mm.) was evident in 38 per cent of the patients with known red blood cell counts, moderate anemia (3 to 4×10^6 per cu. mm.) was evident in 33 per cent and no anemia was present in 30 per cent. (Table XII.) In the eighty patients with known hemoglobin levels, 88 per cent had values between 6 and 15 gm. per cent, while in the seventy-three patients with known hematocrits, 90 per cent had readings between 26 and 45 per cent. (Tables XIII and XIV.) Our findings confirm the conclusions that the presence of anemia (as indicated by a low red cell count) and leukocytosis are valuable laboratory aids in the diagnosis of splenic injury. Many have stressed the importance of roentgenograms for further confirmation of the diagnosis [*4,11,13,16,17*]. Radiologic studies were carried out for sixty-nine patients. Of these patients, 43 or 62 per cent of the results were noncontributory to the diagnosis. Sixteen per cent showed evidence of paralytic ileus, while the characteristic findings of increased density in the left upper quadrant and gastric dilatation occurred in 7 per cent and 6 per cent of the patients, respectively. A few of the patients showed the classic elevation in the left side of the diaphragm, free abdominal fluid and shift of the stomach to the right. The findings of serration of the greater curvature of the stomach and obliteration of the splenic shadow that were stressed by O'Neill and Rousseau [*11*] were found in only two of the patients studied [*6*].

Urinalysis was performed in seventy cases. The only significant finding was hematuria in three of the patients. Although stressed by Wright and Prigot [*18*] paracentesis abdominis was only carried out in three patients. Free blood was aspirated in two patients.

TABLE XIII
HEMOGLOBIN

Hemoglobin (gm. %)	No. of Patients	Per cent of Patients with Known Hemoglobin Values
0 to 5	8	10
6 to 10	33	40
11 to 15	37	46
16 to 20	2	3
Not known	3	...

TABLE XIV
HEMATOCRIT

Hematocrit (%)	No. of Patients	Per cent of Patients with Known Hematocrit Values
16 to 25	6	8
26 to 35	34	47
36 to 45	32	44
Over 45	1	1
Not known	10	...

TABLE XV
DELAY OF SURGERY AND MORTALITY

Time from Injury to Surgery (hours)	No. of Patients	Per cent of Surgical Patients	No. of Deaths	Mortality (%)
0 to 9	44	60	5	11
10 to 19	11	15	1	9
20 to 29	5	7	0	0
30 to 39	3	4	0	0
Over 40	10	14	1	10
Totals...	73	100	7	10

TABLE XVI
RESULTS OF TREATMENT

Treatment	No. of Patients	No. of Deaths	Mortality (%)
Splenectomy.....	73	7	10
No splenectomy.....	10	4	40
Total.....	83	11	13

TREATMENT

The standard treatment for splenic rupture is splenectomy. Seventy-three per cent of our patients underwent surgery, with the majority of them operated on within nine hours from the time of injury. There appears to be no correlation between mortality and the delay of surgery. (Table xv.) Of the ten patients who did not undergo splenectomy, four died giving a mortality rate of 40 per cent; whereas, the mortality rate for the patients who had splenectomy was only 10 per cent. (Table xvi.)

If the arbitrarily set period of only forty-eight hours delay in onset of symptoms is considered a "delayed rupture" of the spleen, then eight of the eighty-three patients (10 per cent) may be included in this category with the delay ranging from forty-eight hours to twenty-two days [12]. Fifty per cent of the procedures performed in this series involved an incision splitting the rectus abdominis muscle on the left side; 21 per cent involved a transverse incision and 16 per cent, a paramedian incision on the left side. Other types performed in a few instances were a subcostal incision on the left side, an incision on the right side of the upper midline and a hockey-stick incision. Sixty-three per cent of the operations lasted between two and three hours. No correlation between the lengths of time taken during surgery and mortality was evident.

POSTOPERATIVE COMPLICATIONS

Of the seventy-three patients who had a splenectomy, 74 per cent recovered without any postoperative complications. Of the nineteen patients in whom complications did develop, 32 per cent had pulmonary atelectasis. Wound sepsis and pneumonia accounted for 16 per cent of the complications. Other disorders are given in Table xvii.

COMMENTS

Various investigators have reported mortality rates ranging from 46 per cent to 92 per cent. As would be expected, the mortality rate was highest in the older age group. In scanning the mortality figures for the sixteen series reported, one is immediately aware of the variability from series to series in this rate. We could find no correlation between the delay in performing surgery and the mortality rate, as there was only one death in the eleven cases in which surgery was performed ten or more hours after the reported accident. The variability in signs and symptoms from patient to patient was evident. They ranged from all of

TABLE XVII
POSTOPERATIVE COMPLICATIONS

Complications	No. of Patients	Per cent of Patients with Complications
Pulmonary atelectasis.....	6	32
Wound sepsis.....	3	16
Pneumonia.....	3	16
Subphrenic abscess.....	2	11
Cardiac arrest.....	2	11
Elevated temperature.....	2	11
Hemothorax.....	1	5
Distension.....	1	5
Pseudocyst of pancreas.....	1	5
Pulmonary congestion and edema.....	1	5
Asthma.....	1	5
Anuria.....	1	5
Paralytic ileus.....	1	5
Gastric dilatation.....	1	5
None.....	54	...

TABLE XVIII
CAUSES OF DEATH IN ELEVEN PATIENTS

Cause	No. of Deaths
Hemorrhage	6
Irreversible shock	3
Other	
Fractured skull	1
Lacerated lung	1
Lacerated liver	1

the expected signs of a ruptured intra-abdominal viscus to no abdominal complaints whatsoever. Nevertheless, pain in the left side of the abdomen with nausea and vomiting may still be considered the splenic rupture syndrome. Pain occurring in the left shoulder was present in only 4 per cent of the patients; this figure was much lower than expected. It must be concluded that the absence of Kehr's sign does not rule out rupture of the spleen, but, when present, it is helpful for focusing attention on the possibility of splenic rupture. Roentgenograms of the abdomen revealed serrations of the greater curvature of the stomach in only two of the sixty-nine patients so studied, thus indicating the relative importance of this finding in diagnosis. (Table XVIII.)

In conclusion it must be stated that the signs and symptoms of a ruptured spleen are variable, and that the diagnosis is frequently obscure immediately following injury. Yet in this age of increasing speeds, violence and tensions, the incidence will increase. Therefore, importance must be attached to any one sign or symptom if early diagnosis is to be made and treatment instituted.

SUMMARY

1. An analysis of eighty-three cases of traumatic rupture of the normal spleen is presented.

2. Male persons outnumber female persons in their susceptibility to this type of injury. In our series 70 per cent of the patients studied were of the male sex.

3. The peak incidence of traumatic rupture of the spleen occurs in the first two decades of life.

4. This injury is more often caused by the blunt type of trauma rather than by the penetrating type. The penetrating wounds, however, cause the greatest mortality.

5. The so-called spontaneous cases of ruptured spleen are most likely due to either a trivial or unrecognized injury or one that occurred some time previous to the rupture.

6. There is a high incidence of associated injuries in persons with a ruptured spleen. In our series, over 75 per cent of the patients had injuries to other organs of the body.

7. Localized pain in the left side of the abdomen together with nausea and vomiting are so frequent in patients with splenic rupture that it might be considered the "splenic rupture syndrome."

8. Leukocytosis and evidence of a declining hemogram are extremely important laboratory findings in the diagnosis of traumatic splenic rupture.

9. The roentgenographic findings are usually noncontributory in the diagnosis.

10. The only satisfactory treatment for traumatic rupture of the spleen is still splenectomy.

REFERENCES

1. BESSEL-HAGEN, F. Beitrag zur Milzchirurgie, *Verhandl. deutsch. Gesellsch. Chir.*, 29: 713, 1900.
2. BLAIN, A., III. The diagnosis and emergency management of ruptured spleen. *Alexander Blain Hosp. Bull.*, 11: 1929, 1952.
3. BOLLINGER, J. A. and FOWLER, E. F. Traumatic rupture of the spleen, with special reference to delayed splenic rupture. *Am. J. Surg.*, 91: 561, 1956.
4. BUSNER, J. W., WALLNER, A. and LEASK, W. H. Roentgen diagnosis of rupture of the spleen by pneumoperitoneum. *J. Internat. Coll. Surgeons*, 20: 338, 1953.
5. BUXTON, J. T. Traumatic rupture of the spleen. *Am. J. Surg.*, 36: 312, 1922.
6. FOSTER, J. M. and PREY, D. Rupture of the spleen; an analysis of twenty cases. *Am. J. Surg.*, 47: 487, 1940.
7. FULTZ, C. T. and ALTEMEIER, W. A. Delayed rupture of the spleen after trauma. *Surgery*, 38: 414, 1955.
8. KNOPP, L. M. and HARKINS, H. N. Traumatic rupture of the normal spleen. *Surgery*, 35: 493, 1954.
9. LARGHERO, Y. P. and GUIRIA, F. Traumatic rupture of the spleen. *Surg. Gynec. & Obst.*, 92: 385, 1951.
10. MANSFIELD, R. D. Traumatic rupture of the normal spleen. *Am. J. Surg.*, 89: 759, 1955.
11. O'NEILL, J. F. and ROUSSEAU, J. P. Roentgenological examination of the abdomen as an aid in the early diagnosis of splenic injury. *Ann. Surg.*, 121: 111, 1945.
12. PARSONS, L. and THOMPSON, J. E. Traumatic rupture of the spleen from nonpenetrating injuries. *Ann. Surg.*, 147: 214, 1958.
13. PERISHO, G. M. and STEINER, M. Traumatic rupture of the spleen. *U. S. Nav. M. Bull.*, 43: 216, 1944.

14. Roettig, L. C., Nusbaum, W. D. and Curtis, G. M. Traumatic rupture of the spleen. *Am. J. Surg.*, 59: 293, 1943.
15. Rousselot, L. M. and Illyne, C. A. Traumatic rupture of the spleen. *S. Clin. North America*, 21: 455, 1941.
16. Wang, C. C. and Robbins, L. L. Roentgenologic diagnosis of ruptured spleen. *New England J. Med.*, 254: 445, 1956.
17. Webb, R. C. Traumatic rupture of the normal spleen with delayed hemorrhage. *Lancet*, 59: 545, 1939.
18. Wright, L. T. and Prigot, T. Traumatic subcutaneous rupture of the normal spleen. *Arch. Surg.*, 39: 551, 1939.
19. Zabinsky, E. J. and Harkins, H. N. Delayed splenic rupture. A clinical syndrome following trauma, 174 cases. *Arch. Surg.*, 46: 186, 1943.

Cancer of the Large Bowel in the Young Adult

ALLAN HALL, M.D. AND ROBERT J. COFFEY, M.D., *Annandale, Virginia*

From the Department of Surgery, Georgetown University School of Medicine, Washington, D. C.

CARCINOMA of the large intestine is usually thought of as a disease primarily affecting patients in the middle and elderly age group; however, it has been shown in a study of patients with gastrointestinal tumors in the military age group that 77 per cent of malignant epithelial tumors occur in the large intestine [*10*]. Except for discussions relating to the disease in childhood and the occasional case reports of its occurrence in young adults and adolescents, the surgical literature contains few studies of colonic cancer in patients of the young age group.

The present study is a review of all cases of proved carcinoma of the large bowel in patients between the ages of sixteen and forty years inclusive, treated during the twelve year period, 1946 through 1957. A clinicopathologic survey of data relating to these patients will be presented with emphasis on diagnosis and prognosis. According to the location of the primary lesion, the study has been divided into a "right colon" group and a "left colon" group. Included as right colon are the cecum, ascending colon, hepatic flexure and transverse colon. The left colon includes the splenic flexure, descending colon, sigmoid, rectum and anus.

INCIDENCE

A total of 718 patients with cancer of the large bowel were seen from 1946 through 1957 and fifty of these were in the sixteen to forty year age group. This is an incidence of 6.9 per cent, which is comparable with other reports [*6,23,27,28*]. Twenty-eight (56 per cent) of the patients were men, while twenty-two (44 per cent) were women. As might be expected, there was a greater incidence with increase in age. In the sixteen to twenty year and the twenty-one to thirty year age groups there were twelve patients (24 per cent). The thirty-one to forty year age group accounted for thirty-eight patients (76 per cent). (Table I.)

There was a total of fifty-one carcinomas of the large bowel in the fifty patients. Nineteen of these were located in the right colon and thirty-two were in the left. (Table II.)

In the general distribution of the lesions, 64 per cent were found in the left colon, a lower percentage than is usually reported [*3,5,6,15, 22,32*]. Twenty-five (50 per cent) of the lesions were located in the sigmoid colon or rectum. It is interesting to note that carcinoma of the transverse colon occurred only in male patients and that sigmoid and rectal cancers were found in equal distribution among men and women.

One patient had a second nonsimultaneous primary malignancy of the colon. This patient was a nineteen year old woman, with rectal carcinoma, who was found to have a primary cecal carcinoma during a laparotomy for intestinal obstruction fifteen months after the initial Miles' resection. Although this is a small series, this one instance of multiple primary cancer of the colon would represent 2 per cent of the group. This incidence is below the 2.9 to 5.8 per cent reported by other investigators [*4,16,20,21,30*].

TABLE I
INCIDENCE ACCORDING TO AGE GROUP

Age Group (yr.)	Male Patients	Female Patients	Total
16–20	0	2	2 (4%)
21–30	8	2	10 (20%)
31–40	20	18	38 (76%)
Total	28 (56%)	22 (44%)	50

TABLE II
LOCATION OF PRIMARY LESION

Location	No.	Per Cent	Male Patients	Female Patients
Right colon				
Cecum	4*	8	2	1
Ascending colon	5	10	4	1
Hepatic flexure	5	10	2	3
Transverse colon	5	10	5	...
Total	19	38	13	5
Left colon				
Splenic flexure	2	4	1	1
Descending colon	4	8	1	3
Sigmoid	12	24	6	6
Rectum	13	26	6	7
Anus	1	2	1	...
Total	32	64	15	17

* One cecal carcinoma was a second nonsimultaneous primary tumor.

TABLE III
SYMPTOMS OF CARCINOMA OF THE COLON

Symptoms	Right Colon (18 patients)		Left Colon (32 patients)		Total (50 patients)	
	No.	Per Cent	No	Per Cent	No.	Per Cent
Abdominal pain	17	94.4	20	62.5	37	74
Rectal bleeding	4	22.2	22	68.7	26	52
Constipation	6	33.3	14	43.7	20	40
Vomiting	7	38.8	8	25.0	15	30
Weakness	8	44.4	7	21.8	15	30
Nausea	6	33.3	7	21.8	13	26
Anorexia	6	33.3	7	21.8	13	26
Diarrhea	1	5.5	11	34.4	12	24

SYMPTOMATOLOGY

There were no patients in this study who were entirely asymptomatic. The duration of symptoms prior to diagnosis ranged from two days to twenty-four months, the average being six months. The location of the tumor made no significant difference in the duration of the symptoms.

No attempt was made in the evaluation of the symptoms to differentiate them as to first symptoms or presenting symptoms, but rather to consider all symptoms present prior to diagnosis. The accuracy of symptom analysis is always debatable because of two major variables: the patient's recollection of symptoms and the ability of the examiner to elicit precise symptomatology. Over-all frequency of symptoms, comparative incidence as to location and grouping of related symptoms are shown in Tables III and IV.

Abdominal pain was the most frequent symptom (74 per cent) of all patients; following in frequency were rectal bleeding (52 per cent) and constipation (40 per cent). Of the right colon group, abdominal pain occurred in 94.4 per cent of patients; second in frequency was weakness; next, were gastrointestinal symptoms.

With carcinoma of the left colon the major symptoms were rectal bleeding, abdominal pain and constipation and these occurred in 68.7, 62.5 and 43.7 per cent of patients, respectively.

A change in the bowel habit consisting of constipation, diarrhea, alternating constipation and diarrhea, tenesmus or any combination of these symptoms was present in 78.1 per cent of patients having carcinoma of the left colon and in 64 per cent of the fifty patients. Upper gastrointestinal complaints such as nausea, vomiting or anorexia were seen in 83.3 per cent

TABLE IV
RELATED SYMPTOMS

Symptoms	Right Colon (18 patients)		Left Colon (32 patients)		Total (50 patients)	
	No.	Per Cent	No.	Per Cent	No.	Per Cent
Change in bowel habits:						
Constipation, diarrhea, alternating diarrhea and constipation, tenesmus	7	38.8	25	78.1	32	64
Upper gastrointestinal disturbances:						
Nausea, vomiting and anorexia	15	83.3	11	34.4	26	52

of patients with carcinoma of the right colon and in 52 per cent of the entire group.

Weight loss was not included in the tabulation of signs and symptoms because of the vagueness and uncertainty of many patients regarding this. It was present, however, in thirteen (72.5 per cent) of the carcinomas of the right side and in thirteen (40.6 per cent) of the lesions of the left side in a total of twenty-six patients (52 per cent) of the entire group.

PHYSICAL FINDINGS

The findings on physical examination have been limited to the palpation of an abdominal mass, hepatomegaly, rectal mass, or a lesion on sigmoidoscopic examination. Physical signs of partial or complete intestinal obstruction such as tenderness, distention and abnormal peristalsis have been excluded from this study.

There was a palpable abdominal mass in each of seven patients (38.8 per cent) with cancer of the right colon, while four masses (12.5 per cent) were detected in patients with cancer of the left side; thus, abdominal mass was present in eleven cases, or in 22 per cent of the fifty patients. Hepatomegaly was noted in one patient with a malignancy of the hepatic flexure and in another with rectal carcinoma.

For the twenty-six lesions in the sigmoid, rectum and anus, a positive sigmoidoscopic examination was present in twenty patients for an incidence of 76.9 per cent of this group. In these same patients a mass was detected on digital rectal examination in fifteen (57.7 per cent). By a combination of sigmoidoscopy and digital examination, the diagnosis could have been made in twenty (40 per cent) of the fifty patients.

LABORATORY STUDIES

Anemia was noted in two thirds (66.6 per cent) of the patients having a malignancy of the right colon, in comparison to 37.5 per cent of those having lesions of the left colon. Almost one half of all the patients in this series were anemic. Examination was made for presence of blood in the stool of thirty patients, with positive results in nineteen (63.3 per cent). The number of positive tests was about equal for patients with malignancy of the right colon and those with malignancy of the left colon. There were eight patients without a history of rectal bleeding who had a positive stool test. If we combine these eight patients with the twenty-six having a definite history of rectal bleeding, thirty-four (68 per cent) of the entire series had either gross or chemically positive evidence of rectal blood loss.

The diagnosis of carcinoma of the large bowel was made radiographically in thirty-five instances (94.6 per cent) of the thirty-seven patients examined by x-ray. A lesion of the ascending colon and another of the sigmoid colon were not diagnosed by barium enema, giving an incidence of 5.4 per cent false negative examinations.

There were nine patients in whom an incorrect diagnosis was made and treatment rendered for gastrointestinal complaints prior to discovery of cancer of the large bowel. (Table v.)

TREATMENT

The operability rate for the entire series was 100 per cent. Curative resections were performed in thirty-three patients for a curative resectability rate of 66 per cent. In eleven patients (22 per cent) the resections were palliative, and no resection was undertaken in the remaining six patients (12 per cent). (Table vi.)

Three deaths occurred in the hospital postoperatively giving an operative mortality of 6 per cent. These deaths occurred in patients receiving palliative treatment only. One was a twenty-two year old man with carcinoma of the ascending colon who died of carcinomatosis twenty-one days after a side to side ileotransverse colostomy bypass. Another was a thirty-three year old man who died ten days after an exploratory laparotomy with carcinomatosis secondary to a primary lesion of the transverse colon. The third patient was a sixteen year old girl (the youngest patient in the series) with a sigmoid carcinoma, who died of widespread abdominal metastases twelve days after an anterior resection and hysterectomy.

Five patients underwent emergency surgical procedures at the time of admission. Intestinal obstruction was present in four, while the fifth patient had a perforated carcinoma of the sigmoid.

PATHOLOGY

Except for squamous cell carcinoma of the anus, all of the tumors were adenocarcinomas. Pathologic grading was reported in only

TABLE V

INCORRECT DIAGNOSES AND TREATMENT PRIOR TO DISCOVERY OF CARCINOMA OF THE COLON

Patients	Age (yr.), Sex and Race	Incorrect Diagnoses	Treatment*	Time Interval before Carcinoma Diagnosed	Location of Lesion
C. W.	36,M,N	Chronic appendicitis	Appendectomy	6 mo.	Sigmoid
D. McK.	36,M,W	Diverticulitis	Medical	6 mo.	Sigmoid
G. R.	39,F,W	Hemorrhoids	Hemorrhoidectomy (barium enema and sigmoidoscopy negative preoperatively)	6 wk.	Sigmoid
A. L.	21,F,W	Appendiceal abscess	Medical	4 wk.	Sigmoid
J. R.	29,M,W	Granuloma inguinale	Medical	4 wk.	Anus
W. W.	27,M,N	Acute appendicitis	Appendectomy	2 wk.	Hepatic flexure
W. B.	27,M,W	Acute appendicitis	Appendectomy	3 wk.	Sigmoid
M. M.	30,F,N	Acute appendicitis and sigmoid diverticulitis	Appendectomy	3 wk.	Sigmoid
Mck. McP.	28,M,N	Acute appendicitis	Appendectomy	6 mo.	Ascending colon

* In all of the appendectomies a normal appendix was removed.

twenty-six of the fifty tumors. None of these were considered to be grade 1 carcinomas. Seventeen of the twenty-six (65.4 per cent) were classified as grade 2 or well differentiated carcinomas. The remaining 34.6 per cent were grade 3 or 4 malignancies. Ten ungraded carcinomas were of the mucinous (colloid) type.

Lymph node metastases were present in 62 per cent of the series. This was most common in patients with metastases of the right colon in which thirteen (72.2 per cent) of the eighteen showed lymphatic involvement.

In one half of the patients there was evidence of serosal involvement, perforation or direct extension into the surrounding tissues. This was evenly distributed among patients with tumors of the right and tumors of the left side.

Visceral metastatic disease characterized by hepatic spread or abdominal carcinomatosis was present in 26 per cent of the patients. The presence or absence of vascular invasion or perineural infiltration was not reported in a sufficient number of cases to make any evaluation possible. (Table VII.)

TABLE VI

TREATMENT

Type	Right Colon	Left Colon	Total
Palliative			
Resection and anastomosis	3	5	8
Colostomy or cecostomy	1	2	3
Combined resection	0	1	1
Exteriorization	0	1	1
Bypass	2	0	2
Explored only	1	0	1
Miscellaneous (subtotal colectomy with ileostomy)	1	0	1
Total	...	...	17
Curative			
Resection and anastomosis	10	10	20
Combined resection	0	9	9
Exteriorization	0	2	2
Miscellaneous:			
Total colectomy with ileostomy	0	1	1
Subtotal colectomy and colostomy	0	1	1
Total	...	...	33

TABLE VII

METASTASES IN PATIENTS WITH CARCINOMA OF THE COLON

Findings	Right Colon (18 patients)	Left Colon (32 patients)	Total (50 patients)
Adenocarcinoma			
Grade 1	0	0	0
Grade 2	2	15	17
Grade 3	2	4	6
Grade 4	1	2	3
Mucinous	7	3	10
Not specified	6	8	14
Lymph nodes positive	13 (72.2%)	18 (56.3%)	31 (62%)
Lymph nodes negative	5 (27.8%)	14 (43.7%)	19 (38%)
Serosal involvement, perforation or direct extension	12 (66.6%)	13 (41.6%)	25 (50%)
Visceral metastatic disease	7 (38.8%)	6 (18.7%)	13 (26%)

TABLE VIII
DEATH OF PATIENTS IN SERIES WITH CARCINOMA OF COLON

Period of Survival after Operation (mo.)	Deaths
0–6	8*
7–12	8
13–18	9
19–24	2
25–30	4
31–36	2
37–42	1
43–48	0
49–54	1
55–60	0
Lost to follow-up	5

* Includes the three postoperative deaths.

TABLE IX
PATIENTS IN SERIES WITH CARCINOMA OF COLON AND STILL LIVING

Patients	Age (yr.), Sex and Race	Primary Site	Survival Time to Date
T. J.	33,W,M	Rectum	34 mos.*
E. M.	34,W,F	Rectum	36 mos.
M. M.	36,W,F	Descending colon	36 mos.
W. B.	39,W,M	Sigmoid colon	46 mos.
J. K.	33,N,M	Splenic flexure	56 mos.
M. McP.	28,N,M	Ascending colon	59 mos.
M. R.	34,N,M	Ascending colon	5 yr., 1 mo.
P. P.	33,W,M	Sigmoid colon	5 yr., 7 mo.
T. H.	39,W,M	Rectum	7 yr., 10 mo.
J. R.	29,W,M	Anus	9 yr., 3 mo.

* Has hepatic metastases.

Only ten patients in this study showed no evidence of lymphatic involvement, tumor penetration of the bowel wall or visceral metastases at the time of treatment. One of these patients had carcinoma of the ascending colon, while the other nine patients had carcinomas located on the left side of the bowel.

Coexistent colonic disease was present in eight patients, or 16 per cent. Four patients had a past history of ulcerative colitis, two had multiple polyposis and the remaining two had multiple polyps.

RESULTS

At the time of this study there are only ten known survivors, or 20 per cent of the total group, and of these one patient has proved hepatic metastases. Thirty-two patients died of colonic cancer; five patients were lost to follow-up; and there were three postoperative deaths. (Table VIII.)

In the group of thirty-five patients who died from carcinoma of the large bowel, twenty-seven (77 per cent) died within two years after operation. The period of survival for patients currently alive ranges from thirty-four months to nine years and three months. Four of these living patients apparently have been free of disease for five or more years. (Table IX.)

COMMENTS

The five year survival rate of patients in this series lends support to the general concept and that expressed in recent articles [*2,11,12,24*] indicating a very poor prognosis for young adults with carcinoma of the large bowel. There are studies, however, in which a more optimistic viewpoint is presented [*6,15,18,19*].

Both in the past and in recent years the majority of statistics gives a five year survival rate ranging from 25 to 50 per cent or more [*1,6–9,13–15,17,22,23,25–29,31,32*]. The reasons for the exceedingly low salvage rate in the present study are difficult to explain, although there are several points worthy of consideration.

The symptomatology, duration of symptoms, resectability rate and extent of resection does not seem to have had a direct influence on the outcome of disease in these young patients since these factors were similar to those noted in older adults with cancer of the large bowel in whom the prognosis appears to be better.

From this study it is suggested that the advanced stage of disease at the time of operation has a significant effect on the end results. Despite the fact that the pathologic examinations were not complete as to tumor grading, vascular involvement or perineural infiltration, 80 per cent of the patients had "unfavorable" lesions as evidenced by lymph node metastases, extension through the bowel wall or distant spread when treatment was instituted.

Cancer of the colon or rectum in young adults appears to be a more rapid and aggressive disease when compared to that usually found in the older patient. The only chance of possibly increasing the number of five year survivals is earlier diagnosis. For this reason the clinician should be suspicious and consider

the likelihood of carcinoma of the large intestine in any young patient presenting with any combination of abdominal pain, evidence of rectal bleeding, a change in bowel habits or upper gastrointestinal complaints.

This is especially important when the symptoms are unexplainable or not typical of any one condition. A thorough physical examination and diagnostic studies directed to the large bowel are mandatory in this situation. The errors in diagnosis and treatment which occurred in this study might have been avoided if the possibility of a large bowel malignancy had been considered preoperatively and a careful exploration had been performed at the time of surgery.

SUMMARY AND CONCLUSIONS

1. A study of fifty young adults with carcinoma of the large intestine has been presented. The symptoms, physical findings and results of diagnostic study are similar to those found in any age group.

2. At the time of treatment 80 per cent of the patients had pathologic findings present which could not be considered favorable for a cure. This seemed to be the major factor of prognostic significance in this study.

3. Cancer of the large bowel in the young adult age group has a poor prognosis due to an accelerated rate of growth and spread. Only four patients (8 per cent) of the fifty are alive five years or more after treatment.

4. Earlier diagnosis is necessary to improve the five year survival rate, and this is dependent on the clinician being aware of and suspecting large bowel malignancy in any patient with appropriate symptoms regardless of his age.

REFERENCES

1. Astler, V. B. and Coller, F. A. The prognostic significance of direct extension of carcinoma of the colon and rectum. *Ann. Surg.*, 139: 846, 1954.
2. Bacon, H. E. and Sealy, W. B. Malignancy of the anus, rectum and sigmoid colon in the young. *Am. J. Surg.*, 45: 339, 1939.
3. Boehme, E. J. and Hanson, P. J. Carcinoma of the colon and rectum. Site of growth of 1,457 lesions. *S. Clin. North America*, 26: 551, 1946.
4. Brindley, G. V. and Rice, J. S., Jr. Multiple primary malignancies of the large intestine. *S. Clin. North America*, 32: 1499, 1952.
5. Broders, A. C., Sr., Phillips, C. and Stinson, J. C. Neoplasms of the large bowel. *S. Clin. North America*, 32: 1511, 1952.
6. Buser, J. W., Kirsner, J. B. and Palmer, W. L. Carcinoma of the large bowel. *Cancer*, 3: 214, 1950.
7. Cattell, R. B. Carcinoma of the colon and rectum. A report of 503 patients treated at the Lahey Clinic 1938–1941, inclusive. *Surgery*, 14: 378, 1943.
8. Colcock, B. P. Prognosis in carcinoma of the colon and rectum. *Surg. Gynec. & Obst.*, 85: 8, 1947.
9. Coller, F. A. and Berry, R. L. Cancer of the colon. *J. A. M. A.*, 135: 1061, 1947.
10. Ehrlich, J. C. and Hunter, O. B., Jr. Tumors of the gastrointestinal tract. *Surg. Gynec. & Obst.*, 85: 98, 1947.
11. Ezzo, J. A. and Mack, R. E. Carcinoma of the colon under the age of forty. *Ann. Int. Med.*, 49: 321, 1958.
12. Fowler, L. H. Malignant epithelial neoplasms, carcinoma and epithelioma occurring in persons under 26 years of age. *Surg. Gynec. & Obst.*, 43: 73, 1926.
13. Gilchrist, R. K. and David, V. C. Prognosis in carcinoma of the bowel. *Surg. Gynec & Obst.*, 86: 359, 1948.
14. Griffin, G. D. J., Judd, E. S. and Gage, R. P. Carcinoma of the right side of the colon: operability, resectability and survival rates. *Ann. Surg.*, 143: 330, 1956.
15. Grinnell, R. S. Results of treatment of carcinoma of the colon and rectum. *Surg. Gynec. & Obst.*, 96: 31, 1953.
16. Grinnell, R. S. The rationale of subtotal and total colectomy in the treatment of cancer and multiple polyps of the colon. *Surg. Gynec. & Obst.*, 106: 288, 1958.
17. Hallstrand, D. E. Carcinoma of the colon and rectum. *Surg. Gynec. & Obst.*, 99: 234, 1954.
18. Johnson, J. W., Judd, E. S. and Dahlin, D. C. Malignant neoplasms of the colon and rectum in young persons. *Arch. Surg.*, 79: 365, 1959.
19. Mayo, C. W. and Madding, G. F. Carcinoma of the rectum and of the rectosigmoid in the young. *Arch. Surg.*, 40: 83, 1940.
20. McGregor, R. A. and Bacon, H. E. Multiple cancers in colon surgery. Report of 162 cases. *Surgery*, 44: 828, 1958.
21. Mider, G. B. Multiple invasive carcinoma of the large intestine. *Surgery*, 20: 744, 1946.
22. Ottenheimer, E. J. and Oughterson, A. W. Observations on cancer of the colon and rectum in Connecticut. *New England J. Med.*, 252: 561, 1955.
23. Postelthwait, R. W., Adamson, J. E. and Hart, D. Carcinoma of the colon and rectum. *Surg. Gynec. & Obst.*, 106: 257, 1958.
24. Rankin, F. W. and Comfort, M. W. Carcinoma of the rectum in young persons. *J. Tennessee M. A.*, 22: 37, 1929.
25. Ransom, H. K. Carcinoma of the colon. *Arch. Surg.*, 64: 707, 1952.
26. Rhoads, J. E. Malignancy of the colon and rectum. *Veterans Admin. Tech. Bulletin*, TB 10-82, January 13, 1952.
27. Shallow, T. A., Wagner, F. B., Jr. and Colcher, R. E. Clinical evaluation of 750 patients with colon cancer. *Ann. Surg.*, 142: 164, 1955.
28. Spear, H. C. and Brainard, S. C. Cancer of the

large bowel. An analysis of 580 lesions. *Ann. Surg.*, 134: 934, 1951.

29. SWINTON, N. W. Recent trends in the treatment of cancer of the colon and rectum. *Am. J. Surg.*, 92: 727, 1956.
30. THOMAS, J. F., DOCKERTY, M. B. and WAUGH, J. M. Multiple primary carcinomas of the large intestine. *Cancer*, 1: 564, 1948.
31. WAUGH, J. M., BLOCK, M. A. and GAGE, R. P. Three and five-year survivals following combined abdominoperineal resection, abdominoperineal resection with sphincter preservation, and anterior resection for carcinoma of the rectum and lower part of the sigmoid colon. *Ann. Surg.*, 142: 752, 1955.
32. WELCH, C. E. and GIDDINGS, W. P. Carcinoma of the colon and rectum. *New England J. Med.*, 244: 859, 1951.

Paralysis of the Vocal Cords after Thyroidectomy

A REVIEW

MATTHEW H. EVOY, M.D., *Seattle, Washington*

From the Department of Surgery, University of Washington School of Medicine, Seattle, Washington.

PARALYSIS of the vocal cords must be regarded as one of the dreadful complications of surgery in benign disease. Injuries to one or both of the recurrent nerves may result in a high percentage of cases in which exposure is poor, hemostasis is ignored or the surgeon confuses competent surgery with fast surgery. There is no implication that nerve injury indicts the surgeon. Paralysis of the vocal cords is the rare but inevitable and probably irreducible complication of the most careful and meticulous operations on and about the thyroid gland. For those who have been unfortunate enough to injure both nerves, little need be said of the sad aftermath; for those who have been spared the worry and anxiety, this treatise may well serve as an alert to and a warning of a most serious complication.

INCIDENCE

The incidence of recurrent laryngeal nerve injury during thyroidectomy has been reported as 0.7 per cent [*1*], 3.9 per cent [*2*], 4.7 per cent [*3*], 5.7 per cent [*4*], 7 per cent [*5*] and 13.3 per cent [*6*]. The incidence of bilateral injury has been found to be 1.5 per cent in a study of 504 patients after thyroid operations [*3*]. In repeat thyroidectomy the risk of nerve injury ranges from 10 to 15 per cent [*7*]. Published figures must be regarded as understating the true percentage of nerve injury because many unilateral injuries are never recognized. The actual number with postoperative cord changes will be known only when every surgical candidate is given a preoperative and postoperative indirect laryngoscopic examination.

Paralysis of the vocal cords may be regarded as a medical disorder in approximately 10 per cent of cases [*8*]. Although the consequences of this paralysis differ in no way from those following surgical recurrent laryngeal nerve injury, the latter phenomenon will receive exclusive consideration in this article.

ANATOMY AND PHYSIOLOGY

The human larynx is an intriguing and perplexing organ that has been the subject of a great deal of investigation. Much of its physiology is controversial; some of its functional anatomy is still in doubt; consequently the understanding of its disturbed anatomy and physiology must of necessity be far from complete. The intrigue of the human larynx has been shown magnificently by the direct laryngoscopic motion pictures by Bell Telephone Laboratories. In these living photographs speed cameras recorded 4,000 frames per second and, with the action slowed 250 times, the muscular function is strikingly shown.

Before considering the laryngeal complications, the pertinent anatomy and physiology of the nerves to the larynx may well be considered. Both recurrent nerves arise in the upper thoracic space, pass in the tracheoesophageal groove and enter the larynx just behind the inferior tubercle of the thyroid cartilage. Very rarely, when the right fourth aortic arch fails to develop normally, the right recurrent nerve will not be recurrent and will pass straight over from the vagus trunk [*9,10*]. The nerves usually lie against the thyroid gland where the capsule is very nondistinct [*11*]. Although the nerve fibers may pass through fissures in the gland, actual penetration of the gland substance is quite contrary to embryologic fundamentals [*12*]. In approximately 73 per cent of nerves,

branching will have taken place below the thyroid cartilage [13]. The branching occurs below the level of the gland in 21 per cent, in the lower two thirds of the gland in 55 per cent, and in the upper third or superior pole level in 24 per cent, and as many as six branches may be present [14]. There appears to be no disparity in the anatomy of the recurrent nerves on either side [15]. The nerve may be anterior or posterior to the inferior thyroid artery [16]. Although it has been held that the original branching of the nerve separates the adductor and abductor components [17], it is also believed that the nerve divides into sensory and motor fibers and that the anterolateral branch is motor [18].

The superior laryngeal nerve usually divides high out of harm's way and the internal or sensory branch passes deep to the superior thyroid artery just below the level of the hyoid bone. The external or motor branch passes downward and inward beneath the insertion of the sternothyroid muscle.

EFFECTS OF NERVE INJURY

Injury to the superior laryngeal nerve is probably relatively rare. If the internal branch is interrupted, a persistent postglutition cough may result, presumably from the entrance of liquid into the respiratory passages, but phonation will probably not be affected. If the external branch is injured, the cricothyroid muscle will be paralyzed and, with the consequent disturbance of the tension and adduction of the vocal cord, the voice may become shallow and tire easily [19].

Trauma to the recurrent nerves produces varied laryngeal disturbances, depending mainly on the number and nature of the interrupted fibers. If all the fibers of a nerve are involved, the ipsilateral intrinsic muscles, except the cricothyroid and interarytenoid muscles, will lose function. Thus, the main abductor, the posterior cricoarytenoid, the main adductor, the anterior cricoarytenoid and the principal cord vibrator, the thyroarytenoid, will be paralyzed. Pure adductor paralysis, with permanent abduction of the cord and consequent loss of voice, is rare. The usual manifestation of recurrent nerve injury, because of the great vulnerability of the abductor fibers [20,21], is paralysis of the cord in the intermediolateral position, and with the passage of time the cord gradually moves into the paramedian or median position [22,23]. If the nerve damage is permanent, the cord will probably never again move lateralward [22,24]. In cases of long standing the cord assumes a bowing deformity with its concavity directed inward, the vocal ligament is somewhat flaccid and the posterior process is tilted forward. The cord assumes a lower position than normally, and the top of the vocal process of the arytenoid cartilage is directed inward and is more prominent than usual [24]. As these gradual changes take place, the voice is found to improve, but the inspiratory airway must of necessity decrease. Although many articles dealing with behavior of paralyzed vocal cords refer to the "cadaveric position," this term has no accurate significance inasmuch as cords in a cadaver may be found in any and all positions [25,26].

The response of the larynx to injury to one or both of the recurrent nerves has brought about a great deal of controversy. If the vocal cord is supplied with adductor and abductor muscles, and the entire recurrent nerve is traumatized, why does not the cord merely remain immobile midway between full adduction and full abduction? Is it because the abductor fibers are more susceptible to injury [20,21]? Or is it because the more phylogenetically primitive function (adductor) is more resistant to trauma [20,21]? Such theories remain unproved to this day, despite monumental investigations and treatises through the years [28–31].

The role played by the uninjured superior laryngeal nerve exerts a great deal of influence in the eventual position of the cord. The action of the cricothyroid muscle, brought about by a tilt of the cricoid cartilage, is adduction and tension of the vocal cord. Thus, a true paralysis of the cord would follow only total interruption of both the recurrent and superior laryngeal nerves. Even then, because of bilateral innervation of the interarytenoid muscle, a rather insignificant muscle play would actually exist. With long standing paralysis of the muscles supplied by the recurrent nerve, it is not unusual to observe atrophy of the cricothyroid muscle, presumably due to absence of antagonistic muscular tension.

PROGNOSIS OF NERVE INJURY

Operative recurrent laryngeal nerve injury has been found to be permanent in roughly three fifths of the cases studied [2,5,6,32,33]. If

spontaneous recovery is to take place, it will usually do so within four weeks and rarely after six months [34]; yet it is possible for evidence of recovery to be delayed for over a year and a half [35]. Nerve damage, even permanent, may not be manifest until several days or weeks after the operation [7].

The recurrent nerves are unusually fragile. It is interesting to note that in experiments on dogs, in which the recurrent nerves were divided, crushed or tied, no evidence of recovery of function was found after a year [36]. It has been shown experimentally that when long segments of the nerves are stripped of their surrounding tissues and allowed to lie exposed, they promptly failed to conduct respiratory stimuli. However, when only a short segment was exposed, function was not impaired [37]. With this extreme vulnerability it is little wonder that some [38,39] have doubted the wisdom of the routine exposure of the recurrent nerves. Yet, one of the most enviable incidences of paralysis of the vocal cords in a large series of thyroidectomies has been reported by a group that exposes the nerve as a routine procedure [1]. A comparison has been drawn in a report of 672 operations; the nerves were exposed in half of the cases, representing exactly one half of the nerves, and in these, 2.1 per cent of the nerves were injured. In the other half, in which the nerves were not exposed, 3.5 per cent of the nerves were thought to have been damaged [33].

Paralysis of the bilateral cord abductors often creates acute suffocation and demands prompt tracheotomy as a life-saving measure. As mentioned, when only one cord is paralyzed, the incident may go unnoticed, especially if voice changes are used as the sole manifestation of nerve injury [6]. The explanation for the trivial change in the voice with unilateral paralysis lies in the compensatory overadduction of the uninvolved cord. In some cases of unilateral injury, however, serious inspiratory distress may develop if the paralyzed arytenoid cartilage falls inward with a forward rotation [7]. The glottic chink is thereby crowded even with wide abduction of the functioning cord.

Expiratory distress is usually absent even in bilateral median paralysis because of the anatomic and physiologic peculiarities of the region. The true vocal cords, when viewed in coronal section, tent up with their convexity facing upward [30]. A stream of air, passing downward along the sides of the ventricles of Morgagni, in effect pushes the cords closer together [40]. This phenomenon has experimental support. Cords in a human cadaver resisted air pressure up to 140 mm. Hg when applied from above, but offered almost no resistance when the pneumatic force was applied from below [41].

TREATMENT

Irreversible damage to the recurrent nerves inactivates one of the most delicate and finely coordinated muscular actions in the body. The thyroarytenoid is the fastest acting muscle known, having a chronaxie in the neighborhood of 0.1 thousandth of a second [42]. Restoration of normal function, then, is completely unattainable without regeneration of damaged nerves. Normal phonation likewise can never be restored. Compensatory phenomena serve to improve the voice, but cannot adjust for a restricted airway.

Surgical relief of postoperative laryngeal stenosis can be highly effective whether it be tracheotomy or direct approaches to the vocal processes. However, a paradox governs the type and magnitude of any corrective operation: The wider the glottic chink the poorer the voice and effectiveness of the cough. Ordinarily, a fixed glottic passage of less then 4 mm. results in a good voice and cough but an inadequate airway. Conversely, an opening greater than 7 mm. gives a good airway but a poor voice and ineffective cough.

Permanent paralysis of one vocal cord almost never requires treatment. In the rare instances of unilateral adductor paralysis, certain operations have been described [43] that intend to bring the abducted cord back to an optimum position for phonation. In bilateral abductor paralysis, treatment of one type or another is always mandatory. Tracheotomy must be regarded as the most urgent treatment and is in fact life saving in many cases. Fortunately, many of those who have had a tracheotomy will eventually be able to dispense with the tube because of the return of function in one or both nerves.

The most natural remedy for recurrent nerve injury would seem to be anastomosis of the nerve ends. This has been so unsuccessful that it is now believed that the procedure is contraindicated. Attempts to anastomose the distal end of the sectioned nerve to other nerve trunks have been only questionably effec-

tive [44–46] and cannot be wholeheartedly recommended.

With the well proved adductor and tensor action of the cricothyroid muscle [47,48], it has been postulated that some relaxation and abduction of a tense median cord might be obtained by deliberate paralysis of this muscle [49]. The clinical effectiveness of such a procedure, intriguing as it seems, probably is quite insignificant [50].

Surgical measures have been concerned mainly with direct approaches to the vocal processes and their supporting structures. The earliest attempts were carried out through anterior laryngotomy, with removal of part or all of the arytenoid cartilage [51–54]. Others carried out the same procedure through a laryngoscope [55,56]. The inevitable trouble from granulation and formation of scar tissue soon discouraged this technic, and submucous dissections were advocated to remove the cartilages and yet obviate this troublesome complication [53,54]. External approach to the cartilages, either to dislocate [57] or extirpate them [58,59], was subsequently advocated with enthusiasm. Then, reportedly for the first time, the transplanting of muscle (omohyoid) was proposed and employed with the intent of rhythmically moving the vocal process outward. The anticipated muscular effect was soon found to be lacking, but a modification in which the arytenoid cartilage was moved outward and secured in the abducting position appeared to effectively improve the airway [60]. A prototype for this operation, in which the cartilage was moved outward and ankylosed in the lateral position through a laryngotomy, had been reported over a decade previously [61].

It must be realized that any surgical enlargement of the glottic chink will adversely affect the voice. Radical removal of one of the vocal processes results in an adequate airway but an explosive cough will be precluded and phonation will be little better than a coarse whisper. It has been claimed that the endoscopic removal of only the posterior extremity of the vocal process leaves a fairly normal, or at least serviceable, voice and still improves the airway [62].

CONCLUSION

The serious consequences of recurrent nerve damage are often permanent and irreversible. Suffocation can be prevented by emergency tracheotomy by an alert surgeon and house staff. Chronic anoxia can be overcome by one of the surgical procedures at our command. But the amazingly and delicately coordinated mechanism of the human larynx will have been lost forever if the nerves fail to regain function.

Knowledge of the anatomy of the nerves and the inferior thyroid arteries is indispensable to the thyroid surgeon. A very impressive maxim concerning the recurrent nerves, "Know where they should be and look where they might be," has been proposed [63]. As long as thyroid disease demands surgical treatment, recurrent nerves will occasionally and inevitably [6,64,65] be injured. The fact that the incidence of nerve injury in one series was nearly twenty times that of another reported series indicates that, with the utmost care in surgical technic and either the demonstration of the nerve or the avoidance of cutting, crushing or tying of any nervelike filament, the integrity of the larynx will only rarely be disturbed.

REFERENCES

1. Lahey, F. H. Routine dissection and demonstration of the recurrent laryngeal nerves in sub-total thyroidectomy. *Surg. Gynec. & Obst.*, 66; 775, 1938.
2. Gisselson, L. Laryngeal paralysis following thyroidectomy. *Acta. chir. scandinav.*, 99: 154, 1949.
3. Mulligan, E. J. Prognosis of laryngeal paralysis following thyroidectomy. *Arch. Otolaryng.*, 35: 732, 1942.
4. Goldman, L. Experiences with thyroidectomy in a thyroid clinic. *J. Clin. Endocrinol.*, 8: 781, 1948.
5. Williams, A. F. Recurrent laryngeal nerve lesions during thyroidectomy. *Surgery*, 43: 435, 1958.
6. Barry, G. M. Injuries to the recurrent laryngeal nerves during thyroid operations. *Proc. Roy. Soc. Med.*, 48: 437, 1955.
7. Muerman, O. H. Vocal cord paralysis following thyroid surgery. *Acta chir. scandinav.*, 101: 360, 1951.
8. Huppler, E. G., McBean, J. B. and Parkhill, E. Causes of vocal cord paralysis. *Proc. Staff Meet. Mayo Clin.*, 30: 518, 1955.
9. Reid, J. The Cyclopedia of Anatomy and Physiology. Anatomical Demonstrations. London. 1847. R. B. Todd.
10. Work, W. P. Unusual position of the right recurrent laryngeal nerve. *Ann. Otol. Rhin. & Laryng.*, 50: 769, 1941.
11. Fowler, C. H. and Hanson, W. A. The surgical anatomy of the thyroid gland with special reference to relations of the recurrent laryngeal nerve. *Surg. Gynec. & Obst.*, 49: 59, 1929.
12. Rustad, W. H. The Recurrent Laryngeal Nerves in Thyroid Surgery, p. 327. Springfield, 1956. Charles C Thomas.

13. Armstrong, W. G. and Hinton, J. W. Multiple divisions of the recurrent laryngeal nerve: an anatomic study. *Arch. Surg.*, 62: 532, 1951.
14. Morrison, L. F. Recurrent laryngeal nerve paralysis: a revised conception based on the dissection of one hundred cadavers. *Ann. Otol. Rhin. & Laryng.*, 61: 567, 1952.
15. Nordland, M. The larynx as related to surgery of the thyroid based on anatomical study. *Surg. Gynec. & Obst.*, 51: 449, 1930.
16. Berlin, D. D. and Lahey, F. H. Dissections of the recurrent and superior laryngeal nerves: relation of recurrent to inferior thyroid artery and relation of superior to abductor paralysis. *Surg. Gynec. & Obst.*, 49: 102, 1929.
17. Morrison, L. F. Recurrent laryngeal nerve paralysis: a revised conception based on the dissection of 100 cadavers. *Ann. Otol. Rhin. & Laryng.*, 61: 567, 1952.
18. Williams, A. F. The recurrent laryngeal nerve and the thyroid gland. *J. Laryng. & Otol.*, 68: 719, 1954.
19. Hirzel, L. F. The superior laryngeal nerve in relation to thyroid surgery. *West. J. Surg.*, 66: 322, 1958.
20. Murtagh, J. A. and Campbell, C. J. The respiratory function of the larynx: relation of fiber size to function in recurrent laryngeal nerve. *Laryngoscope*, 61: 581, 1951.
21. Rosenbach, O. Zur Lehre von doppelseitgen totalen Lähmung des N. Laryngeus inferior. *Breslau ärztl. Ztschr.*, 2: 27, 1880.
22. New, G. B. and Childrey, J. H. Paralysis of the vocal cords: a study of 217 medical cases. *Arch. Otolaryng.*, 16: 143, 1932.
23. Smith, A. B., Lambert, V. F. and Wallace, H. L. Paralysis of the recurrent laryngeal nerves: a survey of 235 cases. *Edinburgh M. J.*, 40: 344, 1933.
24. Clerf, L. H. and Suehs, O. W. Paralysis of the larynx. *Ann. Otol. Rhin. & Laryng.*, 50: 762, 1941.
25. Fein, J. Die Stellung der Stimmbänder an der Leiche. *Arch. Laryng. u. Rhin.*, 11: 21, 1901.
26. Lauffs, J. Über Glottisschluss an der Leiche und seine Bedeutung. Bonn, 1903. P. Bieberstein.
27. Negus, V. E. Certain anatomical and physiological considerations in paralysis of the larynx. *Proc. Roy. Soc. Med.*, 40: 849, 1947.
28. Semon, F. On the proclivity of the abductor fibers of the recurrent laryngeal nerve to become affected sooner than the adductor fibers or even exclusively in cases of undoubted central or peripheral injury or disease of the roots or trunks of the pneumogastric, spinal accessory or recurrent nerves. *Arch. Laryng.*, 2: 197, 1881.
29. Lemere, F. The innervation of larynx: innervation of laryngeal muscles. *Am. J. Anat.*, 51: 417, 1932.
30. Pressman, J. G. and Kelemen, G. Physiology of the larynx. *Physiol. Rev.*, 35: 506, 1955.
31. Negus, V. E. Certain anatomical and physiological considerations in paralysis of the larynx. *Proc. Royal Soc. Med.*, 40: 849, 1947.
32. Till, A. S. Surgery of the recurrent laryngeal nerve. *Proc. Roy. Soc. Med.*, 48: 440, 1955.
33. Riddell, V. H. Injury to recurrent laryngeal nerves during thyroidectomy. *Lancet*, 271: 638, 1956.
34. Jackson, C. Ventriculocordotomy. *Arch. Surg.*, 4: 257, 1922.
35. Hoover, W. B. Bilateral abductor paralysis: operative management by submucous resection of vocal cords. *Arch. Otolaryng.* 15: 339, 1932.
36. Judd, F. S., New, G. B. and Mann, F. P. The effect of trauma upon the laryngeal nerves. *Ann. Surg.*, 67: 257, 1918.
37. Seymour, J. C. and Henry, H. S. Some experimental observations on the innervation of the larynx in cats. *J. Laryng. & Otol.*, 68: 225, 1954.
38. Hawe, P. and Lothian, K. R. Recurrent laryngeal nerve injury during thyroidectomy. *Surg. Gynec. & Obst.*, 110: 488, 1960.
39. Crile, G. Diagnosis and Treatment of Disease of the Thyroid Gland, p. 405. Philadelphia, 1932. W. B. Saunders Co.
40. Rieben, G. Der Spätverlauf der doppelseitigen Stimmelbandlähmung nach Strumektomie. *Chirurg*, 14: 709, 1942.
41. Lauder-Brunton, T. and Cash, T. The valvular action of the larynx. *J. Anat. & Physiol.*, 17: 363, 1883.
42. Best, C. H. and Taylor, N. B. Physiological Basis of Medical Practice, 3rd ed., p. 1315. Baltimore, 1943. Williams & Wilkins Co.
43. Meurman, Y. Mediofixation der stimmlippe bei ihrer vollständigen Lähmung. *Arch. f. Obren-, Nasen- u. Keblkopfb.*, 154: 296, 1944.
44. Frazier, C. H. and Mosser, W. B. Treatment of recurrent laryngeal nerve paralysis by nerve anastomosis. *Surg. Gynec. & Obst.*, 43: 134, 1926.
45. Ballance, O. Some experiments on nerve anastomosis. *Proc. Staff Meet. Mayo Clin.*, 3: 317, 1928.
46. Frazier, C. H. Anastomosis of the recurrent laryngeal nerve with the descendens noni. *J. A. M. A.*, 83: 1637, 1934.
47. Wagner, R. Die Medianstellung des Stimmbandes bei Recurrenslähmung. *Arch. path. Anat.*, 120: 437, 1890.
48. Grossmaun, M. Experimentelle Beitrage zur Lehre von der "Posticuslähmung." *Arch. Laryng. u. Rhin.*, 6: 282, 1897.
49. Freedman, L. M. The role of the cricothyroid muscle in tension of the vocal cords: an experimental study in dogs designed to release tension of the vocal cords in bilateral recurrent laryngeal nerve paralysis. *Laryngoscope*, 66: 574, 1956.
50. Tschiassny, K. Therapeutically induced paralysis of the cricothyroid muscle or its removal in paralytic laryngeal stenosis. *Arch. Otolaryng.*, 65: 133, 1957.
51. Baker, C. H. Report of a case of abductor paralysis with removal of one vocal cord. *J. Michigan M. Soc.*, 15: 485, 1916.
52. Lore, J. M. A suggested operative procedure for the relief of stenosis in double abductor paralysis. *Ann. Otol. Rhin. & Laryng.*, 45: 679, 1936.
53. Hoover, W. B. Bilateral abductor paralysis opera tive treatment by submucous resection of th vocal cords. *Arch. Otolaryng.*, 15: 339, 1932.
54. Dahmann, H. Operative method in bilateral paresis of the abductors (posticus) of the larynx. *Klin. Wchnschr.*, 5: 1783, 1926.

55. Jackson, C. Ventriculocordectomy. *Arch. Surg.*, 4: 257, 1922.
56. Thornell, W. C. Intralaryngeal approach for arytenoidectomy in bilateral abductor vocal cord paralysis. *Tr. Am. Acad. Opbth.*, 53: 631, 1949.
57. Wright, E. S. The Kelly operation for restoration of laryngeal function after bilateral paralysis of the vocal cords: report of three cases. *Ann. Otol. Rhin. & Laryng.*, 52: 346, 1943.
58. Kelley, J. D. Surgical treatment of bilateral paralysis of the abductor muscles. *Arch. Otolaryng.*, 33: 293, 1941.
59. Orton, H. B. Surgical approach for arytenoidectomy in bilateral abductor paralysis of the larynx. *Laryngoscope*, 53: 709, 1943.
60. King, B. T. New and function restoring operation for bilateral cord paralysis: preliminary report. *J. A. M. A.*, 112: 814, 1939.
61. Rethi, A. Die Therapie der durch Posticusparese bedingten Kehlkopfstenose mit Beschrudung von zwei neuen Fällen. *Ztscbr. f. Hals-, Nasen- u. Obrenb.*, 17: 355, 1927.
62. Thornell, W. C. A new intralaryngeal approach for arytenoidectomy in the treatment of bilateral abductor vocal cord paralysis. *J. Clin. Endocrinol.*, 10: 1118, 1950.
63. Piercy, J. E. Discussion of article by Barry, G. M. Injuries to the recurrent laryngeal nerves during thyroid operations *Proc. Roy. Soc. Med.*, 48: 437, 1955.
64. Rustad, W. H. The Recurrent Laryngeal Nerves in Thyroid Surgery, 1st ed., p. 48. Springfield, 1956. Charles C Thomas.
65. Bowden, R. E. M. The surgery of the recurrent laryngeal nerve. *Proc. Roy. Soc. Med.*, 48: 437, 1955.

CASE REPORTS

Replacement of Entire Esophagus by Reversed Gastric Tube Procedure (Heimlich-Gavriliu Operation)

CASE OF LYE STRICTURE OF THE CERVICAL AND THORACIC ESOPHAGUS

BORIS SCHWARTZ, M.D. AND WAYNE W. HALL, M.D., *Paterson, New Jersey*

From the Surgical Services of The Paterson General Hospital, Paterson, New Jersey.

RECENTLY, Heimlich of the United States and Gavriliu of Rumania independently described a physiologic method of replacing or bypassing the esophagus by a reversed gastric tube [*1–3*]. In the Heimlich-Gavriliu operation, a tube is fashioned from the greater curvature of the stomach, brought under the skin of the chest and into the neck and anastomosed to the cervical esophagus. The distal cervical esophagus is closed and allowed to retract into the mediastinum. The operation has been used to bypass or replace the esophagus for carcinomas or benign strictures. If a carcinoma of the esophagus is the cause of the obstruction, the esophagus is resected at a second stage. The following case report is that of a patient with a lye stricture in the cervical and thoracic esophagus who underwent this operation at The Paterson General Hospital. Successful replacement of an esophagus scarred by lye at this high level has rarely been reported.

CASE REPORT

The patient, a twenty-two year old white woman, had been unable to eat solids or liquids since swallowing lye in a suicide attempt in 1953. A gastrostomy was performed at the time of injury; the patient had been nourished by means of a gastrostomy tube for six and one-half years. She was admitted to The Paterson General Hospital in September of 1959, and again in March of 1960 for aspiration pneumonia. Esophagrams revealed a complete obstruction in the cervical esophagus at the level of D_2. (Fig. 1.)

On April 28, 1960, Dr. Heimlich, with members of the staff of The Paterson General Hospital, performed a reversed gastric tube procedure. Through a left subcostal incision, the abdomen was entered and explored. The tail of the pancreas was freed from its bed. The splenic vessels were ligated in the hilus of the spleen, preserving the origin of the left gastroepiploic vessels, and the spleen was removed. An incision was made into the greater curvature of the stomach through the anterior and posterior walls of the antrum and was extended parallel to the greater curvature through both walls of the stomach to a point high in the fundus. The incised edges of the anterior and posterior stomach walls were sutured together forming the gastric tube and reconstituting the remainder of the stomach. (Fig. 2.) An incision was then made in the neck anterior to the left sternomastoid muscle and the cervical esophagus was exposed. The cervical esophagus was transected and the distal end was closed and allowed to retract into the mediastinum. A subcutaneous tunnel was created in the avascular plane anterior to the sternum from the xiphoid to the incision in the neck. The gastric tube was drawn through the tunnel and brought high in the neck, accompanied by its vascular supply, the left gastroepiploic vessels. The reversed gastric tube was anastomosed end to end to the proximal cervical esophagus, restoring gastrointestinal continuity. (Fig. 3.)

On the third day postoperatively, atelectasis of the right upper and middle lobe developed and

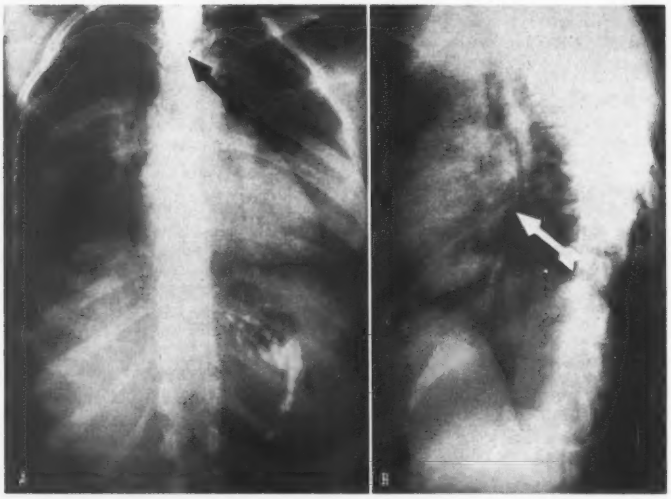


FIG. 1. Preoperative esophagram: A, posteroanterior view. Arrow demonstrates the upper level of almost complete esophageal obstruction with dilatation of the esophagus above. B, lateral view. Arrow demonstrates the stringlike strictured esophagus. A mere trickle of thin barium passes through into the stomach.

cleared following a bronchoscopy. Because Staphylococcus hemolyticus was cultured from the sputum at the time of atelectasis, the patient was placed on penicillin and streptomycin for fourteen days, and Albamycin® for eight days. By the seventeenth day, the pneumonitis secondary to atelectasis had completely cleared.

The patient was maintained on intravenous feedings for the first three days after surgery and

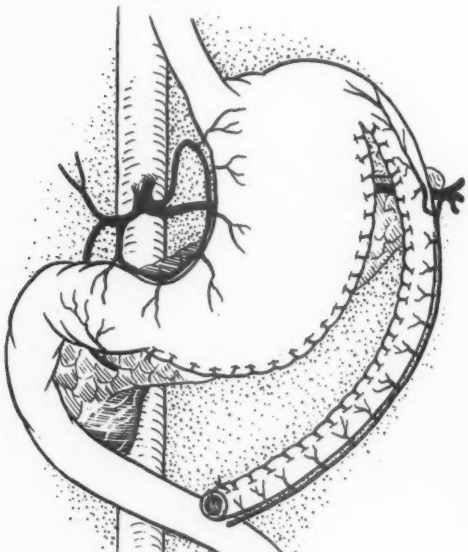


FIG. 2. Construction of the new esophagus (reversed gastric tube) from the greater curvature of the stomach.

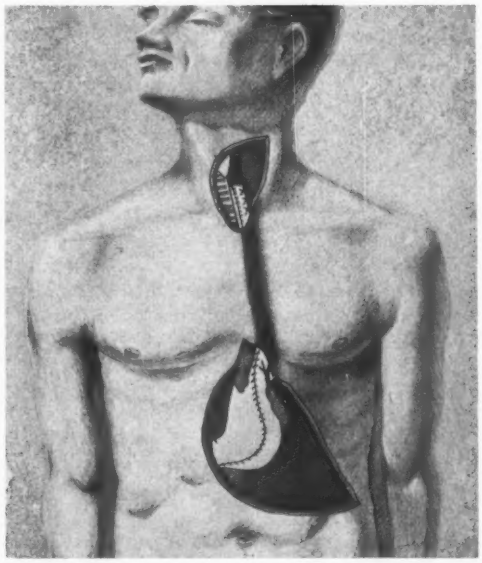


FIG. 3. Completed operation. The new esophagus (reversed gastric tube) has been anastomosed end to end to the cervical esophagus proximal to the obstruction. The distal end of the cervical esophagus has been closed.

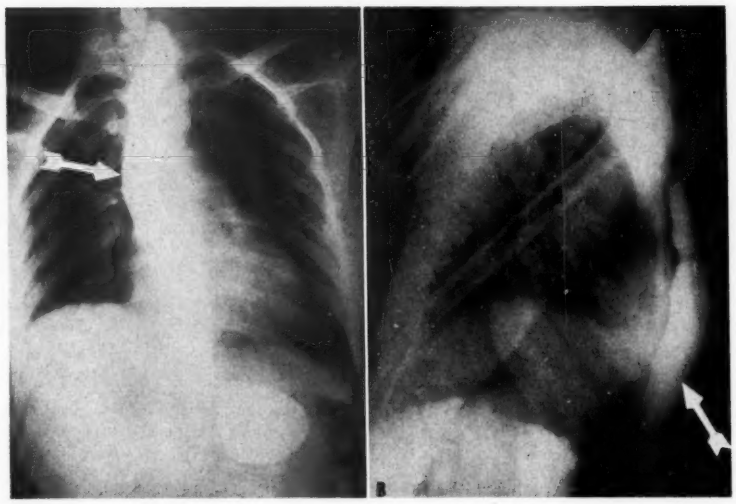


FIG. 4. Postoperative barium swallow. The arrows demonstrate the new esophagus (reversed gastric tube). A, posteroanterior view. The adequate diameter of the reversed gastric tube is evident. B, lateral view. The gastric tube is seen to lie subcutaneously, anterior to the sternum. The tube is seen as it empties into the residual stomach.

then on gradually increased gastrostomy feedings. The gastrostomy feedings were composed of half egg nog and half milk, 1 jar baby meat, 1 jar baby vegetable and a vitamin concentrate. She was given nothing by mouth until the seventeenth postoperative day. From the seventeenth day, the patient was able to progress from a diet of liquids and jello to a soft diet. At the time of discharge, June 14, 1960, after being fitted with a complete set of dentures, the patient was able to eat solid food.

The patient has gained weight well and is able to swallow all foods without difficulty. For a period of one to two weeks she could not lie flat after eating at night because of a "burning" in the area of the new esophagus in the chest. By eliminating the 9 P.M. feeding, this problem was relieved. Since discharge, she has been able to lie flat after meals without difficulty, and eats three good meals a day. (Figs. 4 and 5.)

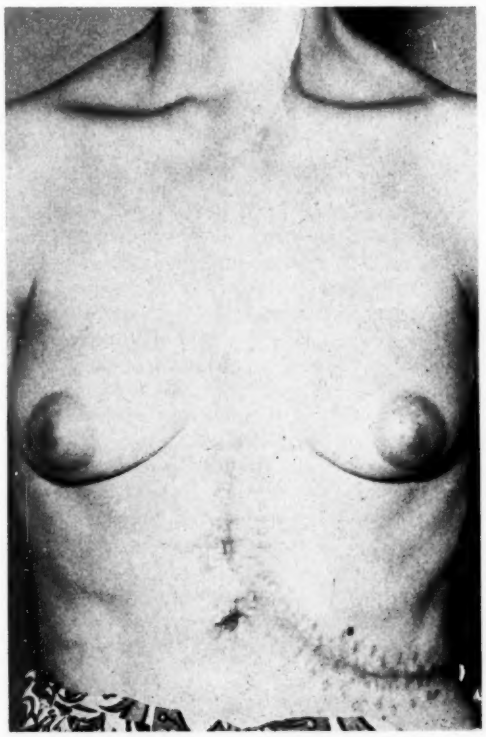


FIG. 5. Postoperative picture of the patient.

COMMENTS

This operation provides a physiologic means of bypassing an esophageal stenosis at any level with minimal morbidity and mortality [*16*]. The reversed gastric tube looks and functions like a normal esophagus. There is no disturbance of intestinal continuity; weight and nutrition are maintained. Four fifths of the stomach remains in the abdomen and retains the normal storage function. Most important

from the patient's point of view, is the fact that she can eat all foods in a normal manner.

Esophageal replacement using jejunum has been used successfully [4–6]. However, there have been difficulties due to inadequacy of the vascular pedicle and ulceration of the jejunum [7,8]. A colon transplant is another means that has been used to replace the esophagus [9–12]. This method was first reported in 1911 [13]. Freshly eaten food in the colon, however, would seem to be an unnatural physiologic condition and may account for deaths that have occurred later. In addition, Nardi [17] recently reported a case of severe peptic ulceration of the colon following colonic replacement of the esophagus and states that there is little evidence that the large bowel is more resistant than the small bowel to digestion by gastric contents. The entire stomach has similarly been used to replace or bypass the esophagus by bringing the stomach into the chest [14]. The complications and deaths resulting from anastomotic leaks into the pleural cavity, and the development of peptic esophagitis make this an unsatisfactory procedure.

Peptic esophagitis does not occur following the reversed gastric tube operation because the antrum, which does not secrete acid, forms the portion of the tube which is anastomosed to the esophagus. The proximal esophagus is, therefore, not subjected to the irritation of regurgitated acid gastric juice. Esophagitis and postsurgical strictures have not been observed in patients followed three to five years after the operation [15]. The advantages in bringing the tube to the neck by the subcutaneous route are: (1) In the event that an anastomotic leak should occur, it is a relatively minor complication which will heal spontaneously in several days or weeks. On the other hand, when this complication occurs following intrathoracic procedures, empyema results, which is almost always fatal. (2) The reversed gastric tube can be constructed and gastrointestinal continuity re-established without entering the chest.

SUMMARY

A twenty-two year old woman with a lye stricture of the cervical esophagus was unable to swallow food or liquids for six years and survived by means of gastrostomy feedings. A reversed gastric tube procedure (Heimlich-Gavriliu operation) which replaced the entire esophagus was performed. The patient now eats a regular diet in a normal manner.

REFERENCES

1. Heimlich, H. J. and Winfield, J. The use of a gastric tube to replace or bypass the esophagus. *Surgery*, 37: 549, 1955.
2. Heimlich, H. J. The use of a gastric tube to replace the esophagus as performed by Dr. Dan Gavriliu of Bucharest, Rumania. *Surgery*, 42: 693, 1957.
3. Gavriliu, D. and Georgescu, L. Esofagoplasti directa cu material gastric. *Rev. stiint. med.*, 3: 33, 1951.
4. Harrison, A. W. Transthoracic small bowel substitution in high stricture of the esophagus. *J. Thoracic Surg.*, 18: 316, 1949.
5. Reinhoff, W. F., Jr. Intrathoracic esophagojejunostomy for lesions of the upper third of the esophagus. *South. M. J.*, 39: 928, 1946.
6. Robertson, R. and Sarjeant, T. R. Reconstruction of the esophagus. *J. Thoracic Surg.*, 20: 689, 1960.
7. Schumacker, H. B., Jr. and Battersby, J. S. Problem of esophageal replacement by jejunum with particular reference to influence upon circulation of staging the division of mesenteric vessels. Experimental study and case report. *Ann. Surg.*, 133: 463, 1951.
8. Watson, W. L. and Cliffton, E. E. Total esophagoplasty using right colon. *Cancer*, 10: 488, 1957.
9. Mahoney, E. B. and Sherman, C. D., Jr. Total esophagoplasty using intrathoracic right colon. *Surgery*, 35: 937, 1954.
10. Neville, W. E. and Clowes, G. H. A., Jr. Reconstruction of the esophagus with segments of the colon. *J. Thoracic Surg.*, 35: 2, 1958.
11. Sherman, C. D., Jr., Mahoney, E. B., Dale, W. A. and Stabins, S. J. Intrathoracic transplantation of the right colon for esophageal reconstruction. *Cancer*, 8: 1198, 1955.
12. Watson, W. L. and Cliffton, E. E. Total esophagoplasty using right colon. *Cancer*, 10: 488, 1957.
13. Kelling, G. Oesophagoplastik mit Hilfe des Querkolon. *Centralbl. f. Chir.*, 38: 1209, 1911.
14. Garlock, J. H. Resection of thoracic esophagus for cancer located above arch of aorta. Cervical esophagogastrostomy. *Surgery*, 24: 1, 1948.
15. Heimlich, H. J. and Gavriliu, D. Discussion on carcinoma of the esophagus. *J. Internat. Soc. Surg.*, p. 780, 1958.
16. Heimlich, H. J. Esophageal replacement with a reversed gastric tube. *Dis. Chest*, 36: 478, 1959.
17. Nardi, G. L. and Glotzer, P. J. Anastomotic ulcer of the colon following colonic replacement of the esophagus. *Ann. Surg.*, 152: 10, 1960.

Jejunal Intussusception

AN UNUSUAL COMPLICATION OF THE USE OF AN INTESTINAL TUBE

ALFRED A. MOSCARELLA, M.D., *New York, New York*

From the Surgical Service, St. Luke's Hospital, New York, New York.

INTUSSUSCEPTION of the small bowel is a rare complication attending the use of the long-limbed intestinal tube. In 1945, Smith [*8*], in an analysis of 1,000 patients intubated with a Miller-Abbott tube, found no instance of intussusception. Since 1945, twelve cases of intussusception implicating intestinal tubes have been reported [*1–7,9*]. Although intussusception following intubation rarely occurs, the attendant mortality and morbidity of this unfortunate complication make an appreciation of its occurrence, symptomatology and pathogenesis essential to those concerned with the use of intestinal tubes.

CASE REPORT

CASE 1. C. P. (No. 401-316), a fifty-year old Negro man, was admitted to the Surgical Service of St. Luke's Hospital on October 5, 1959. On the day of admission a gangrenous appendix was removed. Cultures of the peritoneal fluid taken at the time of operation were reported sterile. He progressed well until the fifth postoperative day when intermittent crampy pain in the abdomen, and slight abdominal distention developed and persistent vomiting occurred. A Miller-Abbott tube was passed into the small intestine. The patient continued to show signs and symptoms of intermittent small bowel obstruction; on the tenth postoperative day his abdomen was explored. During the operation, the point of obstruction was found at the midileum which was bound by a very dense narrow adhesion to the cecum and adjacent appendiceal mesentery. The adhesion was divided and the obstruction relieved.

Postoperatively, the Miller-Abbott tube was left in place. The patient continued to emit small amounts of vomitus, whenever the tube was clamped until the seventh day following the lysis of the adhesions when fluids were tolerated around the clamped tube. Four days later, a Gastrografin® gastrointestinal series showed no evidence of obstruction and the tube was removed. The patient tolerated well a soft diet and was discharged to St. Luke's Convalescent Hospital on the fourteenth postoperative day.

On the seventeenth postoperative day, seven days after the Miller-Abbott tube had been removed, the patient was readmitted to St. Luke's Hospital with the complaint of intermittent

FIG. 1. Film taken one-half hour after ingestion of Gastrografin showing incomplete obstruction of the small bowel. Opaque material in colon and ileum is from gastrointestinal series six days previously.

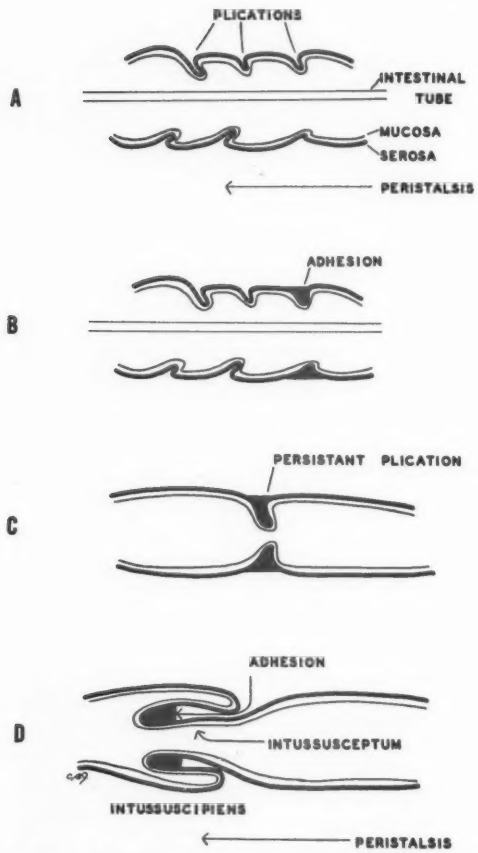


FIG. 2. Pathogenesis of intussusception. A, intestinal plications about intestinal tube; B, formation of adhesion between adjacent serosal surfaces; C, persistent plication; and D, persistent plication forming the lead point of intussusception.

nausea and vomiting following meals. He had no abdominal pain and physical examination of the abdomen revealed nothing unusual. A Gastrografin gastrointestinal series (Fig. 1) showed an incomplete small bowel obstruction just beyond the ligament of Treitz. During operation on the day of readmission an isoperistaltic jejunal intussusception approximately 15 cm. long was found. The leading point of the intussusception was a persistent plication formed by a very dense adhesion which bound the most distal portion of the intussusceptum to the most proximal portion of the intussuscipiens. (Fig. 2D.) The intussusception was reduced, the adhesion divided, and no resection was necessary. The patient progressed without complications and was discharged on the tenth postoperative day. A gastrointestinal series performed three months after discharge demonstrated no abnormalities of the small intestine. He has had no recurrence of symptoms for the past year.

COMMENTS

Of the twelve cases recorded in the literature, five were reports of retrograde intussusception; the remaining seven were instances of isoperistaltic intussusception.

Dunn and Shearburn [2], in 1949, noted the plications of the bowel on the intestinal tube and speculated as to their possible etiologic significance in intussusception of the isoperistaltic type.

McGoon [4], in 1956, called attention to the telescoping of the intestine on the intestinal tube with the consequent formation of multiple plications in the bowel wall. He suggested that normally the peristaltic activity of the bowel over the tube would serve to continually alter the relations of adjacent serosal surfaces of these plications. This action would tend to prevent the adherence of adjacent, inflamed serosal surfaces. However, he then demonstrated that iosperistaltic intussusception occurring after the use of the intestinal tube resulted from the adherence of the inflamed serosal surfaces of adjacent plications in an aperistaltic bowel. Once the serosal surfaces are fused, the resultant plication persists even after the tube is removed; and when peristalsis is resumed, the plication may act as a polyp, forming the lead point of an intussusception. (Fig. 2.)

This case is noteworthy in that symptoms of partial obstruction of the small bowel did not appear until six days after the Miller-Abbott tube was removed. If we assume that the plication leading to the intussusception was present at the time the tube was removed, it seems probable that the return to a more solid diet contributed to the initiation of the intussusception.

Failure to recognize the condition led to a fatal termination in two of the seven reported cases of isoperistaltic intussusception [3,9]. In two of the remaining five cases [1,4], the necessity of small bowel resection was attributed to prolonged delay in diagnosis of the condition.

This case is presented in the hope that it will serve to heighten the awareness of a rare but potentially lethal complication of intestinal intubation, and so lead to earlier diagnosis and prompt intervention in a situation which grows worse with temporization.

CONCLUSIONS

1. If we assume that the intestinal plications once formed, may persist indefinitely, then the possibility of intussusception occurring *at any time* after the removal of the tube must be borne in mind.

2. The high morbidity and mortality attending intussusception following intubation is due primarily to failure to recognize the condition.

3. Early Gastrografin gastrointestinal series in all patients who have been intubated in the past and who have symptoms of obstruction may aid in the prompt recognition of this condition.

REFERENCES

1. Deterling, R. A., Jr., O'Malley, R. D. and Knox, W. Intussusception in the adult, with emphasis on retrograde type. *Arch. Surg.*, 67: 854, 1953.
2. Dunn, D. D. and Shearburn, E. W. Jejunal intussusception: unusual complication of the use of the Miller-Abbott tube. *Surgery*, 26: 833, 1949.
3. Harris, F. I. Intestinal intubation in bowel obstruction. *Surg. Gynec. & Obst.*, 81: 671, 1945.
4. McGoon, D. C. Intussusception: a hazard of intestinal intubation. *Surgery*, 40: 515, 1956.
5. Nichols, H. M. Intussusception occurring after intubation of the small intestine. *Northwest Med.*, 45: 924, 1957.
6. Poppel, M. H. and Brinsley, B. Ileal intussusception as a result of intestinal intubation. *J. A. M. A.*, 169: 1189, 1957.
7. ReMine, W. H. and Pontius, J. G. Intussusception of the small bowel as a complication of a long-limbed intestinal tube. *Proc. Staff Meet. Mayo Clin.*, 33: 451, 1958.
8. Smith, B. C. Experiences with the Miller-Abbott tube. *Ann. Surg.*, 122: 253, 1945.
9. Warren, K. W. and Cattell, R. B. Stenosis of the intestine after strangulated hernia. *Am. J. Surg.*, 75: 729, 1948.

Malignant Mixed Tumor of Parotid Gland

GERALD FINE, M.D. AND CAPT. RICHARD B. MARSHALL, USAF (MC),* *Detroit, Michigan*

From the Department of Pathology, Henry Ford Hospital, Detroit, Michigan.

THE tendency for the seemingly innocent mixed tumor of the salivary gland to recur after local excision is in marked contrast to the rarity of malignancy and metastases. The incidence of malignant mixed tumors has varied with the authors' criteria for both malignancy and mixed tumor. Interchangeable use of the terms mixed tumor and cylindroma and the interpretation of hyalinized areas in the latter and in adenocarcinomas as being indicative of mixed tumor has led to a greater reported incidence of malignancy among mixed tumors than actually exists. Support for this is found in photomicrographs of a number of cases [*10,12*] and our review of slides which, we believe, represent cylindromatous [*5,14*], mucoepidermoid [*5*] and undifferentiated carcinomas [*9*].

Acceptable cases have shown myxochondroid areas together with malignant areas similar to other recognized types of malignant tumor of the salivary gland and only rarely have they maintained a benign histologic appearance in the primary and metastatic sites [*4*].

FIG. 1. Multicystic destructive lesion of femur, simulating a giant cell tumor.

CASE REPORT

H. F. H. (No. 351812), a fifty-two year old white man, was first seen at the Henry Ford Hospital on April 16, 1942 with two subcutaneous nodules at the angle of the right mandible. A right parotid tumor and cervical lymph node had been removed elsewhere four years previously. The duration of the primary tumor was not recorded. The tumor was found to be freely movable and was excised from the muscles of the floor of the mouth. He was well until January, 1943 when he experienced increasing pain in the right thigh and hip. X-ray film (Fig. 1) disclosed a lesion of the right femur and was interpreted as being compatible with a giant cell tumor of bone. Treatment consisted of curettement, filling of the defect with bone chips and administration of 1,800 r to the upper femur. Progressive destruction of the femur with intractable pain and fracture necessitated disarticulation of the right hip in June 1944. In 1945 x-ray film revealed evidence of metastases in the right humerus, multiple ribs and second and third lumbar vertebrae. Intractable pain and anemia progressed until his death in March 1946. There was no evidence of recurrence of the primary tumor. Autopsy was not performed.

PATHOLOGY

The original parotid lesion was removed in three pieces measuring 7 by 3 by 3 cm., 3 by 3 by 3 cm. and 1 by 1 by 1 cm. In the sections studied, the tumor was circumscribed and separated from normal parotid gland by thick, dense, poorly cellular fibrous tissue free of tumor cells. Most of the tumor consisted of cords of uniform, oval, polygonal and spindle cells with intervening thin and occasionally hyalinized

* The opinions and assertions contained herein are those of the authors and are not to be construed as being those of the U. S. Air Force.

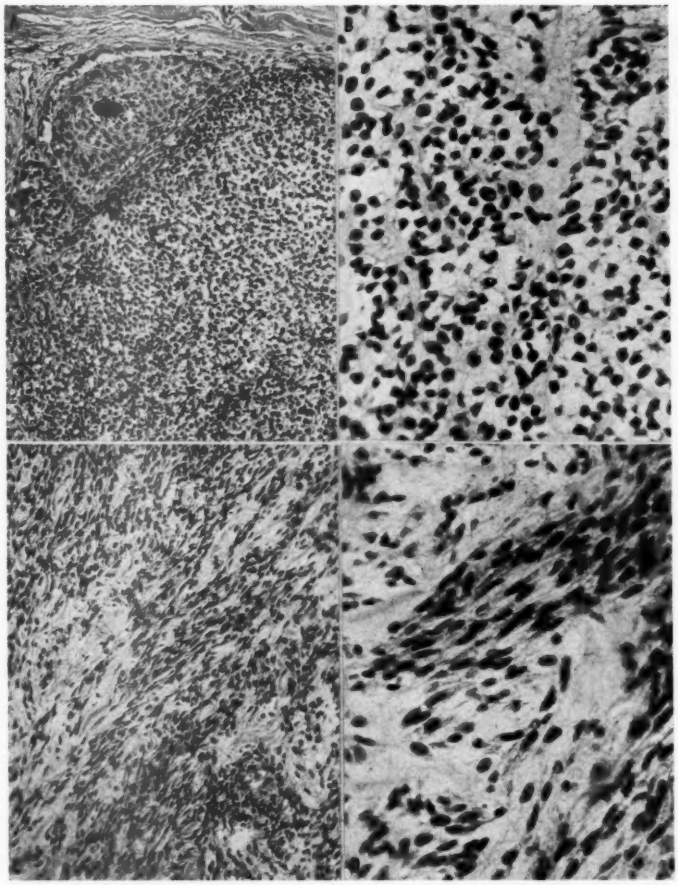


FIG. 2. A, squamous and clear cells of original parotid tumor. B, detail of A, showing trabeculae of cells with clear and reticulated cytoplasm. C, parotid tumor. Area featuring spindle cells with intervening loose fibromyxoid tissue. D, magnification of C. Note resemblance of cells to smooth muscle. (Cells were stained with hematoxylin and eosin. Original magnification of A, × 150; of B, × 400; of C, × 150; and of D, × 400.)

fibrovascular septums dividing the tissue into variously sized masses producing an adenomatous and trabecular pattern. A single cell type was frequently present in a given area, but blending of the cell types was not uncommon. The cytoplasm for the most part was finely reticulated and lightly eosinophilic with well defined cell boundaries, but in some areas it was clear and blended with a lightly basophilic loosely fibrillar stroma. (Fig. 2A and B.) The latter contained stellate cells and small islands of chondroid tissue. Groups of spindle cells simulated muscle tissue but intracytoplasmic fibrils were not seen. (Fig. 2C and D.) Transition of spindle cells to myxochondroid tissue was infrequently seen. Hyperplasia of ductal epithelium with areas of squamous metaplasia obliterated the ductal lumen or extended beyond the confines of the ducts, merging with myxochondroid tissue. (Fig. 3A.) Circumscribed masses of differentiated keratinized squamous cells with intercellular bridges were sparsely distributed in the cellular areas and infrequently merged and blended with myxochondroid tissue and other cells. (Fig. 3B and C.)

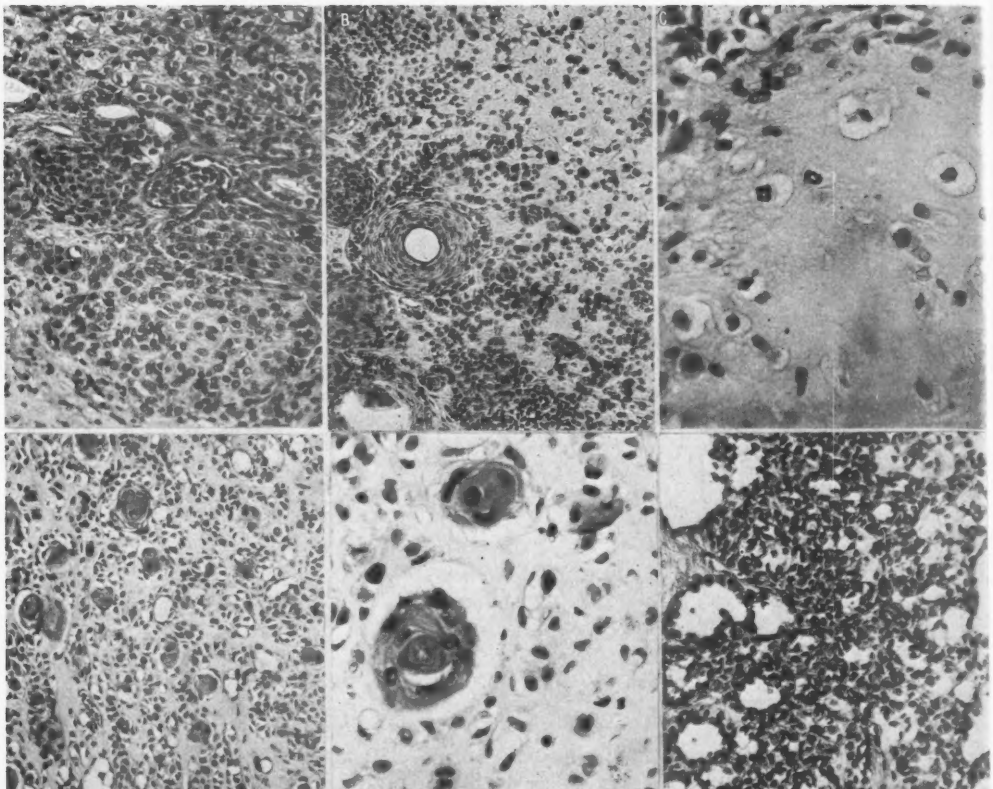


FIG. 3. A, parotid tumor hyperplasia of ductal epithelium obliterating duct lumen and growing outward, merging with the myxochondroid tissue. B, parotid tumor. Squamous and polygonal cells with transformation of latter to myxochondroid tissue. C, detail of B, illustrating chondroid formation. D, metastatic tumor. Squamous and polygonal cell cords with areas of myxochondroid transformation. E, magnification of D. Differentiated squamous and chondroid tissue. F, metastatic tumor. Basal type epithelium with poorly formed tubules. (Cells were stained with hematoxylin and eosin. Original magnification of A, × 300; of B, × 150; of C, × 400; of D, × 150; of E, × 400; and of F, × 250.)

The recurrent tumor was removed in three irregular nonencapsulated pieces not associated with salivary gland, the largest being 4 by 2 by 1 cm. It was similar to the cellular areas of the primary growth with the polygonal and oval cells predominating. No squamous or chondroid tissue was seen, but a small myxoid area was present. There was invasion of skeletal muscle.

The tumor in the femur was more variable than the primary and locally recurrent tumor, having the same pattern and cell types but with more squamous and myxochondroid tissue than the parotid growth. Well differentiated keratinizing squamous cells, thin cords of oval and polygonal cells and clear and spindle cells grew separately or were intimately associated with each other and with myxochondroid tissue. (Fig. 3D and E). Poorly formed tubules of basal type epithelium with infrequent small central foci of squamous metaplasia were also present. (Fig. 3F.)

Very little cellular pleomorphism was noted in any of the material available for study and mitoses were infrequent, averaging 0 to 2 per 50 high power fields. Two lymph nodes from the upper cervical region were negative for tumor.

Alcian blue and periodic acid Schiff stains showed a variable degree of positivity in the

myxochondroid stroma not altered by diastase digestion. The cytoplasm of a moderate number of squamous and chondroid cells were positive with the PAS stain before but not after diastase digestion, and were not stained with alcian blue. Other cells were negative with these stains.

COMMENTS

Studies of morphology and clinical behavior of tumors of the salivary gland over the years has led to a classification which in most instances permits good correlation between histology and behavior of the growth. An exception to this is the mixed tumor. Histologic recognition of the metastasizing potential of mixed tumors is not difficult providing areas similar to other recognized types of malignant tumors of the salivary gland are present. Their absence, however, may make such an observation impossible. Such was the case in the present report in which there was absence of the usual cellular criteria of malignancy in the primary as well as the metastatic tumor. This, in our experience and review of the literature, makes this case unique.

It is tempting to point retrospectively to the cellular areas as malignant, but experience does not bear this out for they have been observed without relationship to metastatic or recurrence rate. In the present case, invasion of skeletal muscle is believed to be of greater prognostic significance than the cellular aberration.

The versatility of the salivary gland epithelium and participation of all of the ductal epithelium with the basal (cylindroid) cells predominating is well illustrated by this tumor. The latter, we believe, are the source of the spindle and basal cells and are partial contributors to the clear cells. Spindle cell and adenomatous patterns are more commonly seen in the myo- and adenomyoepitheliomas, but they have been observed in benign and malignant mixed tumors, [*2,3,4,15*]. Although the myxochondroid differentiation was insignificant in the primary tumor and recurrence, we believe its presence places this growth in the mixed category.

SUMMARY

A case of malignant mixed tumor of the parotid gland with metastases to multiple bones is reported. The mixed character of the tumor was most prominent in the well differentiated, benign appearing femoral metastasis while the primary tumor and local recurrence were predominantly adenomyoepitheliomatous with sparse but well defined myxochondroid components.

Acknowledgment: We wish to thank J. R. McDonald, M.D., Harper Hospital, Detroit for permitting us to study slides from the primary tumor mentioned in the case report.

REFERENCES

1. Ahlbom, H. E. Mucous and salivary gland tumors. A clinical study with special reference to radiotherapy based on 254 cases treated at the Radiumhemmet, Stockholm. *Acta radiol.* (supp.), 23: 1, 1935.
2. Bauer, W. H. and Fox, R. A. Adenomyoepithelioma (cylindroma) of palatal mucous glands. *Arch. Path.*, 39: 96, 1945.
3. Fine, G., Marshall, R. B. and Horn, R. C. Tumors of the minor salivary glands. *Cancer*, 13: 653, 1960.
4. Foote, F. W., Jr. and Frazell, E. L. Tumors of the major salivary glands. Atlas of Tumor Pathology, section IV, fascicle 11. Washington, D. C., 1954. Armed Forces Institute of Pathology.
5. Hudson, R. V., Smith, R. and Smith, C. P. Carcinomatous change in "mixed" salivary gland tumors, with a report of two cases. *Brit. J. Surg.*, 30: 175, 1942.
6. Livingston, S. K. Teratoma (mixed tumor) of the right parotid gland with metastasis to sternum and second rib left; case report. *Am. J. Roentgenol.*, 44: 887, 1940.
7. McFarland, J. Three hundred mixed tumors of the salivary glands, of which sixty-nine recurred. *Surg. Gynec. & Obst.* 63: 457, 1936.
8. McKnight, H. A. Malignant parotid tumor in the newborn. *Am. J. Surg.*, 45: 128, 1939.
9. Montanus, W. P. Mixed tumor of the parotid gland with metastasis: a case report. *Surgery*, 4: 423, 1938.
10. Mulligan, R. M. Metastasis of mixed tumors of the salivary glands. *Arch. Path.*, 35: 357, 1943.
11. New, G. B. and Childrey, J. H. Tumors of the tonsil and pharynx (three hundred and fifty seven cases). II. Adenocarcinomas of the mixed tumor type (seventy-four cases). *Arch. Otolaryng.*, 14: 699, 1931.
12. Olson, G. W. Pulmonary metastases occurring from aberrant mixed salivary gland tumors. Report of three cases and discussion. *Laryngoscope*, 47: 252, 1937.
13. Patey, D. H. The treatment of mixed tumors of the parotid gland. *Brit. J. Surg.*, 28: 29, 1940.
14. Perrin, T. L. Mixed tumor of the parotid with metastases. *Arch. Path.*, 33: 930, 1942.
15. Sheldon, W. H. So-called mixed tumors of the salivary glands. *Arch. Path.*, 35: 1, 1943.
16. Stewart, F. W., Foote, F. W. and Becker, W. F. Mucoepidermoid tumors of salivary glands. *Ann. Surg.*, 122: 820, 1945.

Unusual Skin Metastases from Carcinoma of the Gallbladder

RUSTICO C. TONGCO, M.D., *Cleveland, Ohio*

From the Department of Pathology, Western Reserve University School of Medicine, at Cleveland Metropolitan General Hospital, Cleveland, Ohio.

MALIGNANT tumors may present themselves in a variety of bizarre ways. In some patients, malignant tumors may exist without any clinical symptoms and are found incidentally during surgery or at autopsy [7]. In others, the presence of recognizable metastatic lesions with or without any accompanying clinical symptoms, may be the first indication of the existence of a malignant neoplasm.

Carcinoma of the gallbladder is notoriously a silent carcinoma and often is difficult or impossible to diagnose before operation. The symptoms are usually vague and the presenting signs are difficult to determine and to interpret [2,6]. The case reported herein is unusual in that the first indication of serious disease is the appearance of metastatic nodules in the skin. As far as can be determined from review of the literature this is the third case of carcinoma of the gallbladder with metastasis to the skin to be recorded.

CASE REPORT

CASE 1. The patient, a sixty-nine year old white man was admitted to Cleveland Metropolitan General Hospital on October 10, 1959, because of anorexia and a loss of weight of fifteen pounds during the month prior to admission. Two days before admission he noted multiple subcutaneous nodules in the areas of the chest and abdomen, became alarmed and sought medical advice. The past history revealed that he had been hospitalized fifteen months previously because of anorexia, loss of weight and a mild depressive reaction. At this time the gallbladder was not visualized by oral cholecystogram and the common duct was dilated. Biliary surgery was contemplated but was postponed until later.

Physical examination revealed the following: blood pressure, 84/40 mm. Hg; pulse, 126 per minute; temperature, 37.3°C.; and respirations, 20 per minute. The patient was markedly cachectic and appeared moderately ill. The scleras were not icteric. Multiple hard movable subcutaneous nodules were present in the neck, shoulder, chest and trunk. The largest nodule measured 1.5 cm. in diameter. The skin over some of the nodules in the shoulder region was eroded. The liver was palpable 5 cm. below the right costal margin. The prostate was twice the normal size. The remainder of the results of examination were negative.

Laboratory findings were as follows: hemoglobin, 9.5 gm. per cent; hematocrit, 28 per cent; and the white blood cell count 14,400 cells per cu. mm. with a normal differential. Urinalysis revealed nothing abnormal. The result of examination of feces for occult blood was negative. Determination of direct bilirubin was 2.9 mg. per cent, of total bilirubin, 4.8 mg. per cent. The blood urea nitrogen was 62 mg. per 100 ml. and alkaline phosphatase was 63 Bodansky units. The total protein was 5 gm. per cent with a 1:1 albumin to globulin ratio. The prothrombin time was 72 per cent of normal.

Roentgenograms of the chest, abdomen and the lumbosacral portion of the spine did not show any evidence of metastatic tumor. A subcutaneous nodule was biopsied. This lesion was circumscribed, greyish white, uniformly firm and measured 1.5 by 1.0 by 0.5 cm. Microscopic examination (Fig. 1) revealed sheets and clusters of large polyhedral cells with distinct outlines and abundant pink cytoplasm. The nuclei were large round and hyperchromatic and the chromatin was present as large irregular clumps.

On the fifth hospital day, the patient went into shock and died. The clinical impression was "carcinoma, primary site unknown."

At autopsy the gross findings included the following: the important lesions were confined to the gallbladder area; the gallbladder was distended and filled with greyish white mucous material; and the wall was thickened and patchy, and greyish white firm cauliflower-like growths were distributed over the mucosal surface. The gallbladder was ob-

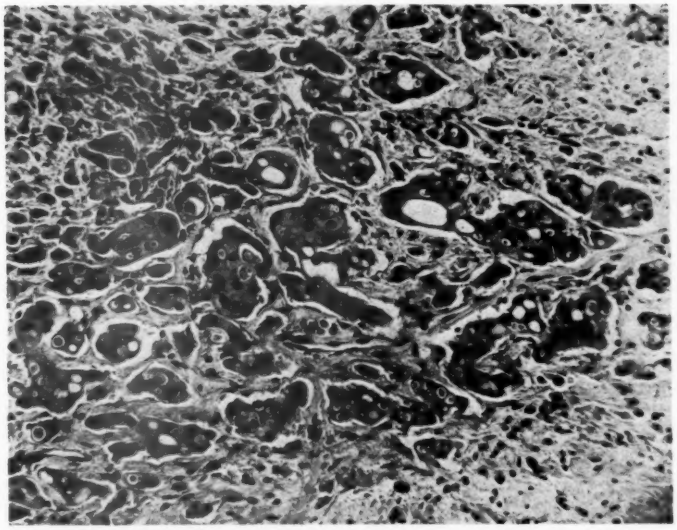


FIG. 1. Microscopic picture of the subcutaneous nodule biopsied.

FIG. 2. Low power magnification of the papillary adenocarcinoma of the gallbladder.

structed by the tumor nodules at the region of the cystic duct. No gallstones were present.

At autopsy (No. A59-288) microscopic examination of a section of the gallbladder (Fig. 2) revealed tumor cells replacing the normal mucosal pattern. The arrangement of the tumor cells in the mucosa were papillary and acinar and the invading cells were arranged in sheets and cords. The cells were moderately large with well defined pink and granular cytoplasm. (Fig. 3.) The shapes varied from columnar and polygonal to spindle. The nuclei were vesicular and contained coarse clumps of chromatin, and the nucleoli were prominent and eccentrically located. Some cells contained intracytoplasmic vacuoles which were demonstrated as mucin by the Mayers' mucin stain. Examples of atypical mitosis were occasionally seen. Metastases were found in the following: the thyroid gland, lungs, liver, pancreas, kidneys and adrenal glands; in the cervical, perihilar, celiac and mesenteric lymph nodes in the pectoralis minor and major and the iliopsoas muscles; and in the skin and sub-

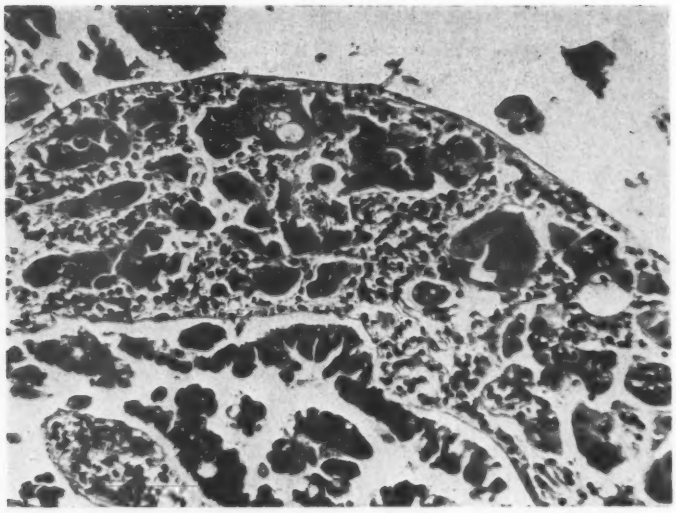


FIG. 3. High power magnification of the carcinoma of the gallbladder.

cutaneous tissues in the chest, neck, shoulders and trunk. These lesions were all greyish white, well circumscribed and uniformly firm. Their histologic appearance was similar to that of the primary tumor, as was the original tumor nodule removed for biopsy.

COMMENTS

During the entire course of this patient's illness, there were no signs or symptoms referable to disease of the gallbladder. Only as the result of a complete examination was an abnormality found; this showed up in the cholecystogram. The first indication of a malignant process was the appearance of nodules in the chest and trunk a few days before the last admission.

Carcinomas of the gallbladder with metastasis to the skin are extremely rare. When metastatic lesions are found in the skin, the primary site is often the skin itself [*16,17*] as in the case of malignant melanomas, lymphomas and carcinomas of the breast. Occasionally, the primary tumor may be in the lung, stomach or kidney [*12,16*]. The gallbladder, however, is considered an unusual source of such metastases and only two other cases have been described previously. Durbeck [*4*], in 1926, reported a case of a carcinoma of the gallbladder which was first evidenced by subcutaneous nodules in the chest and abdomen; Russel and Brown [*11*] reported a case of carcinoma of the gallbladder with metastatic subcutaneous nodules in the thigh. Symmers [*12*] reviewed 298 cases of deep-seated carcinomas in which there were eleven (3.7 per cent) with metastasis to the skin and subcutaneous tissue. Redlick, as quoted by Durbeck [*4*], said that the ratio between carcinomas and skin metastasis was 496:2.

A review of the reports of 9,972 autopsies performed at the Cleveland Metropolitan General Hospital between 1939 and 1959, was carried out and twenty-nine carcinomas of the gallbladder were found. In none of these reports was there any evidence of metastasis to the skin.

Other unusual sites of metastasis of carcinoma of the gallbladder are occasionally encountered. West [*15*], in 1886, recorded a case of a mediastinal tumor which was verified at necropsy as a metastatic lesion of a carcinoma of the gallbladder. Beadles [*1*], in 1897, reported a tumor below the right clavicle, attached to the first, second and third ribs, which originated in the gallbladder. Warthin [*14*] described a carcinoma of the gallbladder with metastasis to the adrenals which simulated Addison's disease clinically. Michaux [*9*] recorded a case of an ovarian cyst which proved to be a carcinoma of the gallbladder. Wakasugi [*13*] described a case of carcinoma of the gallbladder simulating a carcinoma of the rectum. The apparent "carcinoma of the rectum" was a metastatic lesion. McCullagh [*8*] reported a clinically diagnosed

case of twisted ovarian cyst which was discovered at operation to be a large gallbladder with a carcinoma. Rolleston and McNee [*10*] described a case of a carcinoma of the gallbladder in which the first sign of its presence was a metastatic growth in the spine. Foggie and Tudhope [*5*] described a case that was thought to be a primary carcinoma of the left upper lung. Autopsy revealed a "cylindrical cell carcinoma" of the gallbladder. Cappel and Tudhope [*3*] described a case of "lymphosarcoma" which proved to be an adenocarcinoma of the gallbladder.

SUMMARY

An unusual case of carcinoma of the gallbladder that clinically manifested first with subcutaneous nodules is reported and discussed. The review of the literature revealed only two other cases with metastasis to subcutaneous tissue.

REFERENCES

1. Beadles, C. F. Primary carcinoma of the liver (gallbladder) associated with a large tumor on the thoracic wall. *Tr. Path. Soc. Lond.*, 48: 119, 1897.
2. Bockus, H. L. Gastroenterology. Philadelphia and London, 1946. W. B. Saunders Co.
3. Cappel, D. F. and Tudhope, G. R. Unrevealed primary carcinoma of the gallbladder. *Glasgow M. J.*, 6: 185, 1934.
4. Durbeck, K. Über zwei Falle von Krebs mit Hautmetastasen. *Klin. Wchnschr.*, p. 99, January, 1926.
5. Foggie, W. and Tudhope, G. R. Secondary carcinoma of the lung with inconspicuous primary in the gallbladder. *Edinburgh M. J.*, 37: 632, 1930.
6. Liebowitz, H. R. Primary carcinoma of the gallbladder. *Am. J. Digest. Dis.*, 6: 381, 1939.
7. Mattson, H. Carcinoma of the gallbladder. *Minnesota Med.*, 25: 985, 1942.
8. McCullagh, W. Two specimens clinically simulating ovarian cysts. *Proc. Roy. Soc. of Med.*, 18: 47, 1925.
9. Michaux. *Bull. et mém. Soc. chir. de Paris*, 23: 1182, 1907.
10. Rolleston, H. D. and McNee, J. W. Diseases of the Liver, Gallbladder and Bile Ducts, 3rd. ed., p. 691. London, 1929. Macmillan Co.
11. Russel, P. W. and Brown, C. H. Primary carcinoma of the gallbladder. *Am. Surg.*, 132: 121, 1950.
12. Symmers, D. The metastasis of tumors. *Am. J. M. Sc.*, 154: 225, 1917.
13. Wakasugi, K. Zur Kenntnis der sekundaren Neubildungen der Tuben. *Bietr. z. path. Anat. u. z. allg. Path.*, 47: 483, 1909.
14. Warthin, A. S. A case of primary carcinoma of the gallbladder. *Philadelphia M. J.*, 6: 38, 82, 120, 1900.
15. West, S. Cases of mediastinal tumors. *Tr. Path. Soc. Lond.*, 37: 144, 1886.
16. Willis, R. A. The Spread of Tumors in the Human Body. London, 1952. Butterworth and Co., Ltd.
17. Willis, R. A. Pathology of Tumors. London, 1948. Butterworth and Co., Ltd.

Ruptured Pseudocyst of the Pancreas with Associated but Separate Encapsulated Pancreatic Fluid

ARKELL M. VAUGHN, M.D., MICHAEL S. WHITE, M.D. AND JOHN J. RAFFENSBERGER, M.D.,
Chicago, Illinois

From the Department of Surgery, Cook County Hospital, and Vaughn Medical Group, Chicago, Illinois.

RUPTURE of pseudocysts of the pancreas may occur spontaneously from severe pancreatitis or from trauma, both of which may lead to rupture of the capsule or ductal and/or blood vessel systems. When complicated by peritonitis, ruptured pseudocysts of the pancreas carry a high mortality. In a series from Cook County Hospital reported by Meyer et al. [1], in 1949, death occurred in three of four patients who were operated upon for ruptured pancreatic pseudocysts. Rupture of a pseudocyst by needle aspiration was reported recently by Thoroughman and Barrineau [2]. Rupture by this means was first reported by LeDentu [3] in 1865, wherein a pancreatic pseudocyst ruptured while the patient was in the hospital and peritonitis followed. The cyst slowly recurred and several months after the spontaneous rupture, treatment was undertaken by needle aspiration. The patient died several hours later.

We present an unusual case of a spontaneously ruptured pancreatic pseudocyst, with encapsulated pancreatic fluid existing in association with but not connected to the pseudocyst. This undoubtedly is a rare entity since no mention was made of this condition in several extensive reviews on pseudocysts of the pancreas [2,4–6]. In addition, it was thought that a discussion of the characteristics, etiology, symptoms, diagnosis and treatment of pseudocysts of the pancreas *per se* would be in order here to help to illustrate the unusual manifestations and problems encountered in our case.

CHARACTERISTICS AND ETIOLOGY OF PANCREATIC PSEUDOCYSTS

True cysts of the pancreas are rare, but pseudocysts are frequently encountered in this region. These false cysts are saclike spaces, cystlike in nature, but with no epithelial lining membrane. They are composed partially of membranous connective tissue and partly of nearby organs to which they may be attached. Pancreatic pseudocysts contain hemorrhagic and inflammatory exudate in the form of clear or bile-stained fluid, or blood which may or may not contain parenzymes. These cysts rapidly attain a large size. They are usually found within the lesser peritoneal sac. However, they are also found in unusual anatomic locations outside the lesser omental space, yet communicating with a cyst in this space. For example, they were reported to be found extending into the mediastinum, by Edhim [7]; retroperitoneally into the pelvis, by Primrose [8]; and between the layers of the mesocolon, by Judd et al. [9].

In the etiology of pseudocysts of the pancreas two primary factors are inflammation and trauma. In the opinion of Waugh and Lynn [4], however, inflammation seems to be a more frequent forerunner than trauma. This was evidenced in their series of fifty-eight patients with pancreatic pseudocysts, of whom only six attributed the onset of their symptoms to severe trauma.

These pseudocysts usually follow acute pancreatitis, depending upon the degree of destruction of the gland and ductal system. A history

will usually reveal previous severe illness, such as acute pancreatitis, gallbladder disease or necrosis. In fact, according to Whittaker [*10*], most authors report that up to 95 per cent of cases of pancreatic pseudocysts reviewed had previous pancreatitis or gallbladder disease. Severe, penetrating trauma to the epigastrium, nevertheless, may be the primary causative factor in some instances.

SYMPTOMS AND DIAGNOSIS

The symptomatology in pseudocysts of the pancreas is variable. However, development of swelling in the left upper quadrant or in the epigastrium and the presence of a relatively firm, smooth, fixed cystic mass in the epigastrium, with a sensation of fullness or tension, are indicative. In some instances the patient may be asymptomatic until a mass has formed or has greatly enlarged. Pain or signs of pancreatic dysfunction may or may not be present. If the cyst has developed to a large size, anorexia and weight loss may be apparent and are usually concomitant with pancreatic dysfunction or malignancy. If pancreatic destruction has been severe, a mild degree of diabetes and loose, fatty stools may be evident. If regurgitation of bile into the pancreatic ducts after a common duct stone has been an etiologic factor, symptoms of peritonitis will usually be present.

In the diagnosis of pancreatic pseudocysts, fluoroscopic examination with barium meal and barium enema, together with a series of roentgenograms, are invaluable measures. A kidney pyelogram is also a valuable inclusion. These studies will reveal the presence or direction of any displacement of the stomach and colon and the position of the cyst and its locational relationship to the stomach. They will also demonstrate any compression of the duodenum, stomach and colon near the pseudocyst. Widening of the duodenal curve is also a valuable diagnostic sign, demonstrated by roentgen studies. Furthermore, these examinations will, at the same time, be important in ruling out any pathologic lesions of the liver, stomach, spleen and left kidney.

TREATMENT

The management of pseudocysts of the pancreas is primarily surgical, as for all cysts of the pancreas. When surgery is unduly delayed or not carried out, rupture of the pseudocyst is a serious potential, followed usually by peritonitis in which event treatment is, of course, axiomatic. Various operative procedures have been employed according to the surgeon's choice based upon experience, and adaptation of a particular procedure to the individual patient and existing conditions. These include evacuation, complete excision, external drainage by marsupialization, internal drainage by anastomosis to the duodenum, stomach or jejunum, and others.

Some surgeons have found that excision carries a relatively high mortality. External drainage by marsupialization is favored by some surgeons since mortality with this procedure is very low; it is easily accomplished; and is particularly advantageous in large pseudocysts in which the edges of the incised walls can be sutured to the parietal peritoneum. However, there are also disadvantages to the method. For example, because of the necessarily prolonged period of drainage, there is a concomitant loss of fluid, enzymes and electrolytes. Also, because of the irritating quality of pancreatic fluid, the skin often reacts unfavorably, sometimes to the point of erosion or digestion of the skin tissue. Furthermore, according to Whittaker, fistula persists with this method in approximately 50 per cent of the patients and secondary implantation is necessary. Rumore [*11*] pointed out that this is one of the surgical procedures most likely to cause a fistula.

Internal drainage by anastomosis is becoming increasingly favored by many surgeons, and in our experience is the procedure of choice when possible. This method does away with prolonged drainage thus shortening the hospital stay and allowing the patient to leave the hospital in ten to fourteen days. Furthermore, with this procedure drainage is diverted so that the potentiality of skin erosion and multiple operative procedures are avoided. Also, with this method all pancreatic juices can be returned to the intestinal tract to aid digestion.

OPERATIVE TECHNIC OF INTERNAL AND EXTERNAL DRAINAGE

Internal Drainage. In performing internal drainage of a pancreatic pseudocyst, we employ the following technic. The gastrocolic omentum is divided for a sufficient distance to expose completely the anterior surface of the cyst. The stomach is elevated and its posterior wall freed

FIG. 1. Shows the peritoneal cavity opened and the fibrous sac, which had been formed by the irritation of the pancreatic fluid, covering the entire abdominal contents.

from the pseudocyst. A transverse incision 2 to 4 cm. long is made in the cyst wall and the cavity is evacuated by suction.

The jejunum is divided between clamps about 10 to 12 inches distal to the ligament of Treitz. The distal end of the jejunum is brought through an opening in the midportion of the transverse mesocolon and is approximated to the opening in the wall of the pseudocyst. As large an opening as possible is used for the anastomosis. Two layers of sutures are used, employing either plain No. 3–0 intestinal gut for the inner row and No. 3–0 silk for the outer layer, or both layers of No. 3–0 silk interrupted sutures.

The proximal end of the divided jejunum is anastomosed to the distal limb 10 to 12 inches below the pancreatic cystojejunostomy. Here an end to side anastomosis by the Roux en Y procedure is performed, using the same suture material. The opening in the mesentery is closed. The contents of the cyst drain into the jejunal limb in the direction of peristalsis.

External Drainage. For external drainage by marsupialization, we use the following procedure. The abdomen is opened over the area of the pseudocyst, and the cyst is separated from the stomach and colon. The contents of the pseudocyst are aspirated. An incision is made into the pseudocyst for 1 to 2 inches for complete evacuation. The edges of the wall of the pseudocyst are sutured to the edges of the parietal peritoneum. Several Penrose drains are placed in the pseudocyst cavity and the remainder of the abdominal wall is closed around them.

In most pseudocysts the walls will collapse and the cavity will eventually be obliterated. This may require weeks or months. If the marsupialized cyst is a retention cyst and connects with a pancreatic duct, the drainage may change in character and eventually become pure pancreatic juice, and drainage may persist indefinitely. When obstruction to the duct is near the head of the pancreas, large quantities of pancreatic juice may be lost from the body and progressive weight loss and debility may result. In some of our cases it has been found necessary to put suction tubes into the fistula or into the pseudocyst and apply suction to the pseudocyst so that the contents would not get to the skin and could even be fed back to the patient.

The following case shows the difficulty in diagnosis and the problems encountered in management:

CASE REPORT

W. W., a forty-seven year old Negro man, was admitted to Cook County Hospital on May 21, 1959 with a history of generalized abdominal distention and pain of three weeks' duration. This was associated with weight loss of 40 pounds over several weeks. Anorexia had been marked but there were no other gastrointestinal symptoms aside from constipation. The patient had been an extremely heavy consumer of alcoholic beverages for years. Physical examination on admission revealed a chronically ill, emaciated individual complaining of abdominal pain. The abdomen was tensely distended and was dull on percussion. No masses were palpable and tenderness was unremarkable. The impression of the admitting intern was that of ascites with cirrhosis.

Laboratory data revealed hematocrit, 44 per cent and a total protein of 5.7 gm. with an albumin-globulin ratio of 1:1. The remainder of the examination of the liver was within normal limits.

A paracentesis, performed soon after admission, revealed 5 quarts of amber-colored fluid with a specific gravity of 1.015. On microscopic examination many white blood cells were seen. An upper gastrointestinal roentgenographic series demonstrated widening of the duodenal loop. A peritoneoscopy was performed two weeks after admission and 4,000 cc. of fluid was released. The examiner noted many adhesions within the abdominal cavity and a red cystic mass in the upper part of the abdomen. A biopsy of the liver was interpreted

FIG. 2. Demonstrates the massive fibrinous adhesions between the omentum and loops of bowel. This condition existed throughout the abdomen.

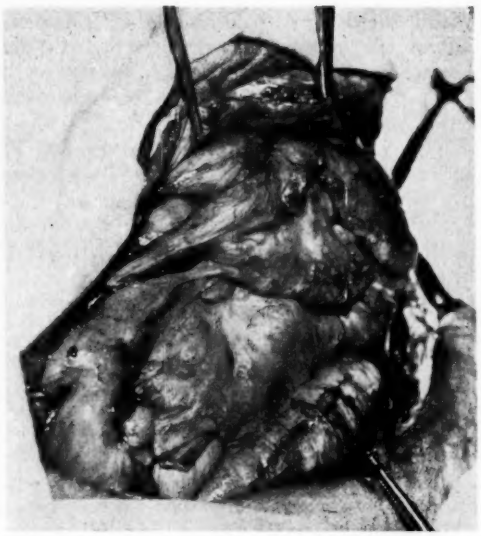


FIG. 3. Shows the bowel and abdominal contents covered with membrane. The pseudocyst can also be seen in this view.

as revealing early Laennec's cirrhosis with fatty changes.

The patient's condition was one of unrelenting abdominal pain, so severe as to require increasing doses of narcotics for relief. Surgical consultation was called, with a diagnosis of carcinoma of the pancreas in mind, and operation of an exploratory nature was decided upon.

On June 25, 1959 the patient was prepared for surgery, with transfusions of whole blood, plasma and amino acids. Because of his poor condition surgery was carried out under local infiltration anesthesia. A supraumbilical transverse incision was made, and revealed chronic peritonitis. The small bowel was matted together with thick adhesions and all of the peritoneal surfaces were a dull red. A large cystic mass was found between the anterior abdominal wall and the loops of small bowel. This sac contained 2,000 cc. of brown turbid fluid. (Figs. 1 and 2.) Further exploration revealed a pancreatic pseudocyst presenting through the gastrocolic omentum. (Fig. 3.) This was opened, and fluid identical to that found in the first cyst was evacuated. The amylase content of fluid from each source was 8,192 Somogyi units. The inside of the pancreatic pseudocyst was smooth and no communication with the cystic mass in the abdominal cavity could be found.

The pancreatic pseudocyst was drained with a Pezzer catheter, and it was possible to shell the major portion of the secondary pseudocyst away from the abdominal wall and the adherent loops of bowel. Biopsy from the pancreatic pseudocyst wall revealed fibroconnective tissue infiltrated with hemorrhagic extravasation and inflammatory cells. Drainage from the tube gradually diminished over a period of two months; contrast media injected into the tube on several occasions showed a gradual diminution in the size of the cyst.

On August 15 feculent drainage was noted coming from the fistulous tract. Injection of the tract with Lipiodol outlined the transverse colon. Drainage gradually ceased and it was thought that spontaneous fistulization into the colon must have occurred. The patient was discharged from the hospital on August 25, 1959. When seen six months after operation, no drainage was present, the patient had gained weight and he was apparently well.

COMMENTS

The pseudocyst of the pancreas in the case we have presented, ruptured spontaneously, spilling its contents over the peritoneal cavity, setting up a fibrinous peritonitis. It appeared as though the abdominal contents were completely erupted. The leakage of fluid which occurred and the general peritoneal reaction which ensued was similar to that found in the lesser bursa during an attack of acute pancreatitis. The fluid became encapsulated by inflammatory reaction and connective tissue

within the peritoneal cavity in the same manner in which pseudocysts are formed in the lesser omental sac.

The patient was considered to be in the terminal stage of disease. Therefore, when, at exploratory operation, the ruptured pseudocyst and encapsulated pancreatic fluid were discovered, it was thought that external drainage by marsupialization was the least hazardous procedure for this seriously ill and debilitated patient.

SUMMARY

An unusual case is presented of a ruptured pancreatic pseudocyst with associated but separate encapsulated pancreatic fluid. Peritonitis was also present and the problems encountered in management of the patient are described.

Treatment of pseudocysts of the pancreas is primarily surgical and the advantages and disadvantages of various surgical procedures for this purpose are discussed. The operative technics of internal and external drainage as employed by us are described.

It is emphasized that when surgery of a pancreatic pseudocyst is delayed or not carried out, rupture is highly potential, and when complicated by peritonitis, this carries a high mortality.

REFERENCES

1. Meyer, K. A., Sheridan, A. I. and Murphy, R. F. Pseudocysts of the pancreas. *Surg. Gynec. & Obst.*, 88: 219, 1949.
2. Thoroughman, J. C. and Barrineau, C. E. Spontaneous rupture—pseudocyst of pancreas. *Am. Surgeon*, 23: 831, 1957.
3. LeDentu. Rapport sur l'observation precedents. *Bull. Soc. anat. de Paris*, 10: 197, 1865.
4. Waugh, J. M. and Lynn, T. E. Clinical and surgical aspects of pancreatic pseudocysts: analysis of fifty-eight cases. *Arch. Surg.*, 77: 47, 1958.
5. Brilhart, K. and Priestly, J. T. Pseudocysts of the pancreas. *Am. J. Surg.*, 81: 151, 1951.
6. Warren, W. D., Marsh, W. A. and Sandusky, W. R. An appraisal of surgical procedures for pancreatic pseudocyst. *Ann. Surg.*, 147: 903, 1958.
7. Edhim, P. Mediastinal pseudocyst of the pancreas. Case report and discussion. *Gastroenterology*, 17: 96, 1951.
8. Primrose, A. Pancreatic cysts and pseudocysts. *Surg. Gynec. & Obst.*, 34: 431, 1922.
9. Judd, E. S., Mattson, H. and Mahorner, H. R. Pancreatic cyst. *Arch. Surg.*, 22: 838, 1931.
10. Whittaker, L. D. Traumatic pancreatic cysts. *Illinois M. J.*, 113: 1, 1958.
11. Rumore, P. C. External pancreatic fistula. *Illinois M. J.*, 107: 24, 1955.

Multiple Primary Carcinoma of the Uterus

REVIEW OF THE LITERATURE AND REPORT OF A CASE

ALLAN G. W. McLEOD, M.D. AND DENIS CAVANAGH, M.D., *Miami, Florida*

From the Department of Obstetrics-Gynecology of the University of Miami School of Medicine, Jackson Memorial Hospital, Miami, Florida.

MULTIPLE primary cancer is not an uncommon finding. The subject has been discussed by many authors and much has been written about it since it was first reported by Billroth [*1*]. In contrast, squamous cell carcinoma of the cervix associated with adenocarcinoma of the endometrium is very rare. It has been described by Warren and Gates [*2*] who listed the references to six cases from the German literature. Three cases were reported by Brown and his co-workers [*3*] and several other authors have each reported a case [*4–10*]. Liu and Meigs [*11*] noted one case and mentioned in addition, two cases of squamous cell carcinoma of the cervix combined with adenocarcinoma *in situ* of the endometrium. Dituri and Williams [*12*] reported a case in which a leiomyosarcoma was found in the corpus in addition to adenocarcinoma of the endometrium and coexisting squamous cell carcinoma of the cervix. The latter cervical lesion has been reported in combination with adenocarcinoma arising in an adenomyoma of the corpus [*13*]. Pearson [*14*] reported a case and also referred to three cases in the early German literature and one in the Italian literature not listed by Warren and Gates. An additional five cases in the French and German literature are mentioned by Springer and Denisart [*15*] but the references are unsatisfactory. DeFillipo and Meieres [*16*] in the Spanish literature cited one case and reported three additional cases. One case of squamous carcinoma of the cervix has been reported as occurring simultaneously with carcinosarcoma of the endometrium [*17*].

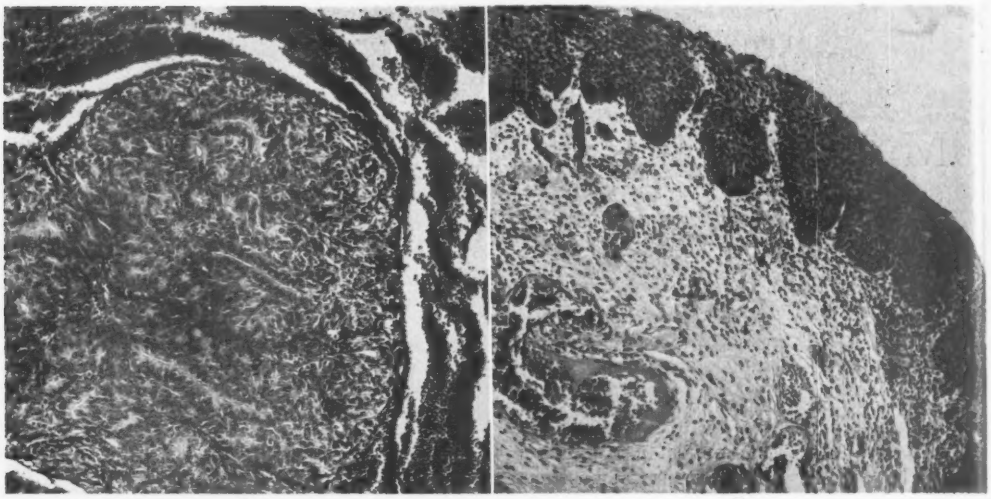
Thus a careful review of the literature reveals that invasive squamous carcinoma of the cervix, with coexistent adenocarcinoma of the endometrium, has been recorded only fourteen times in the English literature, if the case of carcinosarcoma is excluded. In addition nine acceptable cases have been reported in the German and French literature, four in the Spanish and one in the Italian literature. The simultaneous occurrence of squamous cell carcinoma *in situ* of the cervix, and adenocarcinoma of the endometrium has to our knowledge been reported in only one case [*18*].

In reviewing our hospital records over the past eight years, we could find only three patients who had been discharged with this diagnosis. After we reviewed the tissue sections, one of these cases was rejected because it was believed that the epithelial lesion represented dysplasia rather than carcinoma *in situ* of the cervix. The second case was rejected because we could not be certain that the adenocarcinoma was of endometrial rather than endocervical origin. It is our opinion, however, that the following case fulfills the criteria for the diagnosis of this rare type of double primary cancer.

CASE REPORT

Mrs. E. McC., a seventy year old Negro woman, para 6, gravida 6, was admitted to the Jackson Memorial Hospital on May 31, 1960 with a complaint of postmenopausal vaginal bleeding of four months' duration. The bleeding lasted two to three days at a time and recurred at frequent intervals. Her menopause had occurred at the age of forty-one years. The patient gave a history of arthritis for ten years and hypertension for two years. She had also been treated medically for a "gallbladder upset." In 1955 she had a simple mastectomy for what is said to have been "benign disease" in the left breast. She gave no family history of cancer but one of her sisters had diabetes mellitus and one brother had tuberculosis.

Physical examination revealed the patient to be somewhat obese. Her blood pressure was 150/100



A B

FIG. 1. A, adenocarcinoma of the endometrium; B, intraepithelial carcinoma of the cervix. Original magnification of A and B, × 115.

mm. of Hg. A roentgenogram of the chest showed some cardiac enlargement. A two hour postprandial blood glucose was 113 mg. per cent; serum urea nitrogen 12.2 mg. per cent; hemoglobin 12.4 gm. per cent; hematocrit 40 per cent. Her serologic examination was negative and urinalysis revealed no abnormality.

A vaginal cytologic smear was reported as negative (Papanicolaou Class 1) but diagnostic curettage was carried out on June 2, 1960. A pelvic examination performed at the time of dilatation and curettage revealed a normal marital introitus. Bartholin's glands, Skene's ducts and the urethral orifice appeared normal. There was a first degree cystocele and a small rectocele. The vaginal mucosa appeared healthy apart from a small ulcer on the left lateral wall about 3 cm. from the introitus and measuring 1.0 by 0.5 cm. in diameter. The cervix was parous in type and clean. The uterus was anteverted, firm, and enlarged to the size of an eight weeks' gestation. A uterine sound was passed to a distance of 10 cm. and spongy endometrial tissue (3 ml.) suggestive of endometrial carcinoma was obtained on curettage. Punch biopsies of the cervix and the vaginal lesion were taken.

The pathologic diagnoses (S/7370/60) were adenocarcinoma of the endometrium (Fig. 1A), endometrial polyps, squamous cell carcinoma *in situ* of the cervix (Fig. 1B) and fibroepithelial polyp of the vagina.

A cone biopsy of the cervix was taken on June 8, 1960. At that time the cervix was noted to take Schiller's stain well and no "positive" areas were noted. This specimen revealed chronic cervicitis with focal atypia (S/7370/60). Total abdominal hysterectomy and bilateral salpingo-oophorectomy was performed on June 13, 1960. Neither pelvic nor periaortic lymph nodes were palpably enlarged at the time of the operation.

The patient made a good recovery from surgery and was discharged from the hospital in good condition on June 20, 1960. Pathologic examination of the specimen removed at operation showed no residual intraepithelial carcinoma of the cervix but it did reveal residual adenocarcinoma of the endometrium, some adenomyosis and several small leiomyomas (S/7652/60).

At follow-up examination on August 4, 1960 the Papanicolaou smear was still negative. A small amount of granulation tissue was noted at the vaginal vault. This was cauterized with silver nitrate and triple sulfa cream prescribed for local application. At her next visit the vault was almost healed. Subsequent monthly clinic visits have revealed no evidence of recurrence and her Papanicolaou smears have been negative to date.

COMMENTS

Intraepithelial carcinoma of the cervix, in association with adenocarcinoma of the endometrium, has been reported only once previously. The reasons for the paucity of such cases are probably as follows: (1) Intraepithelial carcinoma of the cervix is a diagnosis which has not been made frequently until the last decade. (2) Patients who develop adenocarcinoma of the endometrium are usually of

postmenopausal age while the mean age for women with intraepithelial carcinoma of the cervix is under forty years. (3) There may be some host factor which predisposes some women to adenocarcinoma of the endometrium while protecting them from carcinoma of the cervix. This latter is an attractive hypothesis. It may explain the relatively small number of cases reported in which invasive carcinoma of the cervix is combined with endometrial carcinoma despite the fact that these lesions exist so commonly independently.

It is difficult to evaluate the situation statistically, however. At the University of Texas—M. D. Anderson Hospital, 2,311 patients with carcinoma of the uterine cervix and 460 patients with adenocarcinoma of the endometrium were seen over the period March 1944 to August 1958. This material was reviewed by one of us (D. C.) and in not a single patient were these lesions found to coexist. On further analysis, however, it was believed that only one or two cases could be expected to occur among this number of patients on the basis of chance.

It seems likely that cervical carcinoma is in some way associated with coitus rather than parity [*19*] and certainly a higher occurrence rate is evident in the lower socio-economic groups [*20*]. On the other hand, adenocarcinoma of the endometrium is more likely to be associated with endocrine disturbances [*21,22*]. In this latter respect it seems to us that although Speert [*23*] found that 9 per cent of his patients with adenocarcinoma of the endometrium had been treated previously with irradiation for bleeding in association with benign disease, their underlying endocrinologic disturbance rather than the irradiation *per se* may have been responsible for the ultimate development of adenocarcinoma.

One further factor which suggests the mutual incompatibility of these tumors is the relatively increased incidence of endometrial adenocarcinoma in Jewish women [*24*] and nuns [*25*]. It is our opinion that these epidemiologic differences are worthy of further consideration. In these differences may lie new clues to the prevention and more effective treatment of cervical and corpus cancer.

The cervical lesion in our patient was found initially because it has become our practice to biopsy the cervix when investigating postmenopausal bleeding, even when no cervical lesion is evident. It is also worthy of note that the vaginal cytologic smear in this case was negative. This emphasizes the importance of diagnostic curettage in the investigation of women with postmenopausal bleeding even in the presence of a negative Papanicolaou smear. When possible, an endometrial biopsy should be done although if this is negative, diagnostic curettage must still be carried out.

SUMMARY

1. The literature on multiple primary cancer with reference to the coexistent occurrence of squamous cell carcinoma of the cervix and adenocarcinoma of the endometrium is reviewed.

2. A case of intraepithelial carcinoma of the cervix occurring simultaneously with adenocarcinoma of the endometrium is reported.

3. Possible explanations for the paucity of reports is discussed and the possibility of these tumors being mutually antagonistic is considered.

4. The necessity for diagnostic curettage in the investigation of postmenopausal bleeding, even in the presence of a negative Papanicolaou smear, is stressed. The value of multiple punch biopsies of the cervix, even in the absence of a visible lesion, is emphasized.

REFERENCES

1. Billroth, C. A. T. Chirurgische Klinik, p. 258. Wein, Berlin, 1879. Quoted by Warren and Gates [*2*].
2. Warren, S. and Gates, O. Multiple primary malignant tumors: a survey of the literature and a statistical study. *Am. J. Cancer*, 16: 1358, 1932.
3. Brown, L. B., Latour, J. P. A. and Dodds, J. R. Multiple primary malignant tumors involving the female genital tract. *Am. J. Obst. & Gynec.*, 73: 127, 1957.
4. Bonney, V. Uterus showing squamous cell carcinoma of the cervix and adenocarcinoma of the body. *Proc. Roy. Soc. Med.*, 7: 227, 1914.
5. Major, R. H. Multiple primary malignant tumors; with report of a case of carcinoma and sarcoma in the same individual. *Bull. Johns Hopkins Hosp.*, 29: 223, 1918.
6. Goldstine, M. T. Squamous cell carcinoma of cervix; adenocarcinoma of fundus of uterus. *Am. J. Obst. & Gynec.*, 30: 137, 1935.
7. Counsellor, V. S. and Butsch, W. L. Double malignant tumors of uterus. *Am. J. Obst. & Gynec.*, 31: 628, 1936.
8. Maliphant, R. G. Multiple primary cancers of uterus. *J. Obst. & Gynaec. Brit. Emp.*, 50: 59, 1943.
9. Embrey, M. P. Multiple primary carcinomata of

uterus. *J. Obst. & Gynaec. Brit. Emp.*, 60: 904, 1953.

10. Jackson, J. R. Simultaneous adenocarcinoma of the endometrium with squamous cell carcinoma of the cervix uteri. *Canad. M. A. J.*, 77: 956, 1957.
11. Liu, W. and Meigs, J. V. Radical hysterectomy and pelvic lymphadenectomy; a review of 473 cases including 244 for primary invasive carcinoma of the cervix. *Am. J. Obst. & Gynec.*, 69: 1, 1955.
12. Dituri, B. and Williams, P. F. Multiple malignant tumors of uterus; report of case. *Am. J. Obst. & Gynec.*, 66: 685, 1953.
13. Smith, C. H. and Masson, J. C. Carcinoma in adenomyoma of uterus and squamous-cell epithelioma of cervix; report of case. *Proc. Staff Meet. Mayo Clin.*, 14: 476, 1939.
14. Pearson, H. L., Jr. Multiple carcinomata of uterus. *South. M. J.*, 40: 728, 1947.
15. Springer, A. and Denisart, P. Cancer total ou cancer double de l'uterus. *Gynéc. et obst.*, 52: 294, 1953.
16. DeFillipo, R. A. and Meieres, A. Carcinomas simultaneos de endometrio y cuello uterino. *Semana méd.*, 108: 234, 1956.
17. Lisa, J. R., Pack, S. T. and Gioia, J. Composite endometrial carcinosarcoma, leiomyofibroma and infiltrating cervical epidermoid carcinoma in same uterus. *Am. J. Obst. & Gynec.*, 63: 1162, 1952.
18. Mulla, N. Carcinoma in situ of the cervix and adenocarcinoma of the endometrium; report of a case. *Am. J. Obst. & Gynec.*, 79: 370, 1960.
19. Heins, H. C., Jr., Dennis, E. J. and Pratt-Thomas, H. R. The possible role of smegma in carcinoma of the cervix. *Am. J. Obst. & Gynec.*, 76: 726, 1958.
20. Wynder, E. L., Cornfield, J., Schroff, P. D. and Doraiswami, K. R. A study of environmental factors in carcinoma of the cervix. *Am. J. Obst. & Gynec.*, 68: 1016, 1954.
21. Ingram, J. E. and Novak, E. Endometrial carcinoma associated with feminizing ovarian tumors. *Am. J. Obst. & Gynec.*, 61: 774, 1951.
22. Mansell, H. and Hertig, A. T. Granulosa-theca cell tumors and endometrial carcinoma. A study of their relationship and a survey of 80 cases. *Obst. & Gynec.*, 6: 385, 1955.
23. Speert, H. and Peightal, T. C. Malignant tumors of the uterine fundus subsequent to irradiation for benign pelvic conditions. *Am. J. Obst. & Gynec.*, 57: 261, 1949.
24. Ober, W. B. and Reiner, L. Cancer of the cervix in Jewish women. *New England J. Med.*, 251: 555, 1954.
25. Gagnon, F. Contribution to the study of the etiology and prevention of cancer of the cervix of the uterus. *Am. J. Obst. & Gynec.*, 60: 516, 1950.

Complete Lumbosacral Dislocation

REPORT OF A CASE

RICHARD K. WHITE, M.D., *Allentown, Pennsylvania*

COMPLETE lumbosacral dislocation is rare. An investigation of the literature for the past several years revealed no similar instance. The severity of the dislocation together with multiple associated injuries presented an interesting problem in care and management.

CASE REPORT

A forty-three year old Negro woman was admitted to the hospital following an automobile accident in December 1956. In addition to the complete lumbosacral dislocation as shown in Figure 1, she sustained multiple lacerations of the left arm and both legs. There were fractures of the third to the tenth ribs, inclusive, on the left side, a fracture of the distal end of the left clavicle, a compound fracture dislocation of the right ankle and a compound fracture of the left tibia.

Following admission to the hospital she was treated initially for shock, pain and hemorrhage. The patient had no movement of her legs and no sensation below her knees. Reduction of the dislocation was accomplished in bed under spinal anesthesia. During the performance of a spinal tap, what appeared to be pure blood ran out through the needle but, after a short time elapsed, the flow became less thick and it was assumed that the needle placement was in the dural canal. Manipulative reduction was carried out by traction in the supine position. Both hips and knees were flexed across the operator's shoulders while the compounded extremities were supported by attendants and traction was executed downward and forward.

FIG. 1. Roentgenogram showing lumbosacral dislocation.

FIG. 2. Roentgenogram after reduction.

The result of roentgenographic examination following the reduction is shown in Figure 2. Admittedly, the reduction does not appear to be complete but no further attempts were made to improve the status.

The neurologic deficit of the patient following recovery from anesthesia was paralysis of the motor and sensory nerves below the level of the knee joint. Bladder and bowel incontinence were also present. The patient was cared for on a Stryker frame with pelvic traction. She became somewhat recalcitrant in the traction and this was removed after ten days. Because of her many difficulties, the fractures of the lower extremities were treated by the closed plaster technic, without debridement or any other care except gross positioning. Approximately six months after injury she regained control of her bladder and bowel and had no further difficulty with these functions.

Two years following injury there was complete union of both fractures and all wounds were healed. Control of the bladder and bowel had been maintained and a complete neurologic deficit below her knees had persisted. She had begun to ambulate with the use of braces.

COMMENT

This rather unusual interesting case allowed us to question again the concept of lumbosacral instability. No external apparatus was used in this instance; in fact, the traction itself was discarded shortly after reduction. The need for lumbosacral fusion was not demonstrated by the patient's subsequent course, either in changes in her x-ray picture or in symptoms in her back.

SUMMARY

A case of complete lumbosacral dislocation is presented. This case was associated with multiple injuries complicating the problem. It demonstrates a continued stable entity without the need of additional fusion. In addition, the associated injuries were treated on a nondefinitive basis which was accompanied by a good result. While these methods are not intended to be recommended, they do demonstrate what can be done under unusual circumstances.

Recurrent Volvulus of the Cecum Associated with Failure of Rotation of the Intestine in an Adult

HARRY V. ARMITAGE, M.D., *Chester, Pennsylvania*

From the Department of Surgery, Taylor Hospital, Ridley Park, Pennsylvania.

INTESTINAL obstruction secondary to anomalous rotation of the intestine is usually thought of as a problem of infancy or childhood. Perhaps this is because the most frequent form of anomalous rotation is failure of descent of the cecum with suspension of the midgut from a pedicled mesentery. This results in duodenal obstruction by bands extending laterally from the cecum, and frequently in volvulus of the entire small intestine. By its very nature, this form of obstruction tends to occur early in life. However, when all forms of anomalous rotation are considered, the onset of intestinal obstruction is delayed until adult life more frequently than is generally realized. Gardner and Hart [1], in reviewing 105 cases of anomalies of intestinal rotation causing obstruction, found that in 52 per cent of their series obstruction occurred in the newborn or that symptoms dated from a few days after birth. Thus, in approximately one half of the cases in this series, obstruction was delayed until adult life. Dott [2] studied thirteen cases in which obstruction occurred and he reported that symptoms began a few days after birth in six patients, at the age of seventeen in one, during adult life in five and during old age in one. Dott calls attention to the fact that volvulus of the small intestine has its highest incidence in early life, and volvulus of the ileocecal segment is the typical lesion of later life.

It is surprising that, after the Ladd operation for duodenal obstruction and volvulus of the small intestine, there is not a tendency for the volvulus to recur throughout life. Gross [3] reported only one instance of recurrent volvulus in 156 cases. He suggests that adhesions resulting from the first operation may tend to fix the bowel and prevent recurrence of the volvulus. Since it is well known that volvulus often appears after an operation that produces adhesions, this explanation does not seem to be satisfactory. Perhaps freeing of the cecum from its fixed position beneath the liver unmoors the anchoring point necessary for volvulus of the intestine.

Anomalous rotation of the intestine may exist without causing any symptoms and, of course, the incidence is impossible to estimate. Of forty-eight cases that Dott collected, thirty-five were discovered accidentally and apparently caused no symptoms. Recurrent episodes of abdominal pain in adults may possibly be attributed to twisting and spontaneous release of the twisted segment of the bowel. It is believed that the lifelong, recurrent abdominal pain in the case being presented was probably the result of torsion of a mobile segment of the bowel. In this case of nonrotation the cecum was found to have twisted through 180° in a clockwise direction at the first operation, and through 360° in a counterclockwise direction at the second operation. The cecum was found first in the left lower quadrant and subsequently in the right upper quadrant.

FIRST ADMISSION

A forty-nine year old white woman was admitted to the hospital on July 7, 1955. Approximately four hours before coming to the hospital she experienced sudden colicky pain in the region of the umbilicus. There was no nausea nor vomiting. During her entire life she had had recurrent episodes of intermittent,

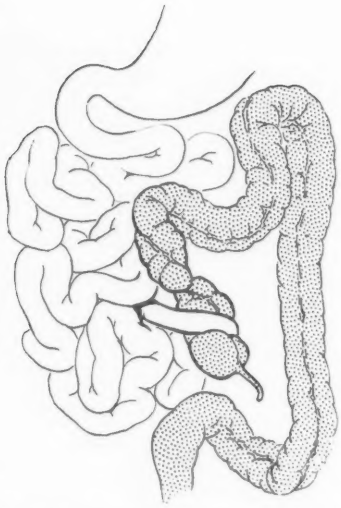


FIG. 1. Volvulus of cecum with nonrotation of intestine. Condition found at first operation.

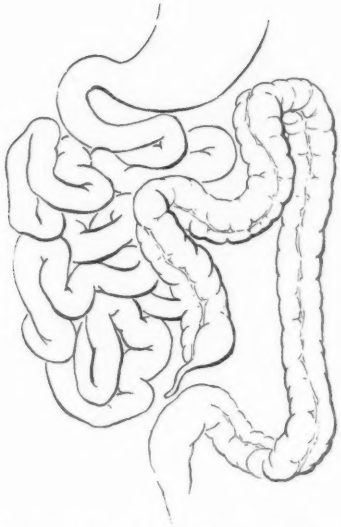


FIG. 2. Derotation of volvulus. Cecum allowed to remain in left lower abdomen. Appendix was removed but this is not indicated here.

lower abdominal pain. The present episode of pain was the most prolonged and the most intense that she had suffered.

Examination revealed a middle aged woman of short stature who appeared in no acute distress. Her abdomen was slightly obese but was not distended. Tenderness and rebound tenderness were present in both lower quadrants, being more marked on the right. Rectal examination showed tenderness in the rectal vault. A diagnosis of acute appendicitis was made and an operation was performed on the night of admission.

OPERATIVE FINDINGS AND PROCEDURE

A right McBurney incision was made, and when the peritoneal cavity was entered, slightly distended loops of small intestine were encountered, but the cecum could not be located. It was believed that possibly the cecum was undescended. Accordingly, the incision was enlarged and the subhepatic area explored. Failing to find the cecum in this location, the rest of the abdomen was explored, and the cecum was discovered in an ectopic position in the left lower part of the abdomen. The cecum was found to share a common mesentery with the small intestine and to have undergone volvulus through 180°. (Fig. 1.) Derotation was readily accomplished and the cecum was delivered out of the wound. The appendix, which was grossly normal, was removed, the cecum was allowed to return to its original position, and the wound was closed. (Fig. 2.)

COURSE

On the second postoperative day the patient vomited several times and complained of cramplike abdominal pain. However, by the third postoperative day she had no complaint and an enema produced effective results. Barium x-ray study of the gastrointestinal tract carried out a week after the operation showed a failure of rotation of the midgut with the small intestine lying on the right side of the abdomen and the large intestine lying on the left side. The cecum was reported as lying in the left lower quadrant. (Fig. 3.) Histologic examination of the appendix showed slight infiltration of the muscular layer with neutrophils. This finding was considered as consistent with either early appendicitis or volvulus. The patient was discharged from the hospital on the eighth postoperative day in good condition.

SECOND ADMISSION

The patient remained symptom free until September 23, 1955. On this date she was awakened from her sleep by severe cramplike abdominal pain. She was again admitted to the

FIG. 3. Nonrotation of intestine. Barium meal study. Cecum lies in left lower quadrant. Entire small intestine lies to the right.

hospital. Examination at this time showed her to be in acute distress. The abdomen was tender throughout with maximal tenderness in the right upper quadrant. Peristalsis was hyperactive and occurred in rushes accompanied by colic. The patient vomited several times after admission. X-ray films of the abdomen revealed distended loops of small bowel in the right upper quadrant, and was reported as showing no gas in the colon. In retrospect, a small amount of gas could be seen in the cecum which was now lying in the upper abdomen. A diagnosis of intestinal obstruction was made and the patient was prepared for operation.

OPERATIVE FINDINGS AND PROCEDURE

A right paramedian incision was made in the midabdomen. The peritoneal cavity was found to contain an increased amount of clear fluid. There were multiple adhesions in the region of the terminal ileum binding it to its mesentery and to the cecum. The cecum and terminal ileum were found to have rotated through 360° in a counterclockwise direction. (Fig. 4.) The volvulus was derotated and the adhesions of the terminal ileum separated. The intestinal current was demonstrated to pass into the transverse colon and the incision was closed.

COURSE

The patient made a good recovery. The nasogastric tube which had been inserted preoperatively was removed on the third postoperative day at which time the patient had three copious watery bowel movements in succession. She was discharged from the hospital on the eighth postoperative day.

Approximately one month after discharge from the hospital the patient complained of a sudden sharp abdominal pain lasting a few

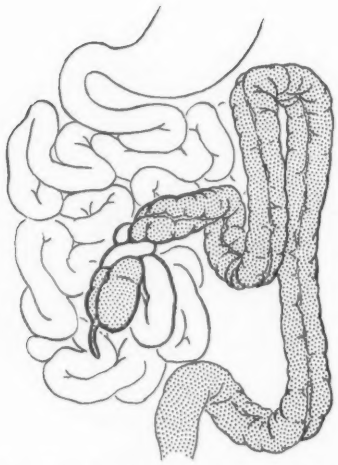


FIG. 4. Condition found at second operation. Cecum has rotated in opposite direction. Appendix had been removed at first operation and should not have been shown.

minutes. The pain vanished as suddenly as it had occurred, and since then she has had no further pain.

COMMENTS

Although the findings were not entirely typical of appendicitis when the patient was seen on the first occasion, it was believed that there was an acute surgical condition present and immediate operation was thought to be the safest course to pursue. A more careful history might have led to a more accurate diagnosis, for later a member of the patient's family recalled that some years before she had been x-rayed in a diagnostic clinic and informed that "part of her bowel was missing."

At the time of the second admission it was believed that the intestinal obstruction was the result of either volvulus or adhesions. Ideally, decompression with a long intestinal tube and preparation for bowel resection might have been desirable, but fear that strangulation obstruction might exist made early surgical intervention seem mandatory.

The adhesions resulting from the first operation might have been instrumental in producing the second more complete episode of volvulus. The ileum was adherent to its mesentery and may have acted as a fixed point for the development of complete volvulus.

At the present time the patient is symptom free and further surgical treatment is not being considered. However, if there is any recurrence of obstructive symptoms, an attempt to correct the basic problem will be made. Resection of the mobile segment of bowel with ileo transverse colostomy has been recommended by Brown and Ross [4] and Williams et al. [5]. As an alternative, the cecum and ascending colon could be sutured to the descending colon as recommended by Findlay and Humphries [6].

SUMMARY

1. A case of recurrent volvulus of the cecum associated with nonrotation of the midgut is presented.

2. It is postulated that the recurrent, lifelong abdominal pain present in this patient was the result of chronic cecal volvulus.

3. Two episodes of volvulus occurred and it is believed the second more complete volvulus was related to the adhesions produced by the first operation.

4. Surgical correction of the anomalous condition has not been necessary in this case but will be recommended if there is recurrence of obstructive symptoms. It does not seem likely, however, that further trouble should develop since five years have elapsed since the last operation.

REFERENCES

1. GARDNER, C. E. and HART, D. Anomalies of intestinal rotation as a cause of intestinal obstruction: report of two personal observations. Review of one hundred and three reported cases. *Arch. Surg.*, 29: 942, 1934.
2. DOTT, N. M. Anomalies of intestinal rotation: their embryology and surgical aspects with report of five cases. *Brit. J. Surg.*, 11: 251, 1923.
3. GROSS, R. E. The Surgery of Infancy and Childhood. Philadelphia, 1953. W. B. Saunders Co.
4. BROWN, R. B. and ROSS, D. Congenital abnormalities of intestinal rotation and mesenteric attachment. A cause of intestinal obstruction in the adult. *Ann. Surg.*, 134: 88, 1951.
5. WILLIAMS, L. F., HUGHS, C. W. and BOWERS, W. F. Volvulus of the cecum associated with malrotation of the intestine in an adult. *Arch. Surg.*, 81: 593, 1960.
6. FINDLAY, C. W. and HUMPHRIES, G. H. Congenital anomalies of intestinal rotation in the adult. *Surg. Gynec. & Obst.*, 103: 417, 1956.

Idiopathic Massive Hemobilia

L. Kraeer Ferguson, M.D. and Moreye Nusbaum, M.D., *Philadelphia, Pennsylvania*

From the Department of Surgery, Graduate School of Medicine and Graduate Hospital, University of Pennsylvania, Philadelphia, Pennsylvania.

The cause of massive hemorrhage from the upper gastrointestinal tract is often difficult to find and even more difficult to control. We have held that blood which appears in vomitus or in the gastric aspirate should localize the bleeding point in that part of the gastrointestinal tract between the lower esophagus and the ligament of Treitz. In patients in whom a previous gastric operation has been performed, this area must be extended downward to include the upper jejunum. We wish to present a case in which massive hematemesis occurred a month after a pancreatoduodenectomy for carcinoma of the head of the pancreas. The bleeding was found to come from the left hepatic duct. The problem of controlling such bleeding and our method of solving it will be presented:

CASE REPORT

U. W., a fifty-six year old Negro man, was admitted for the first time to the Graduate Hospital of the University of Pennsylvania on May 1, 1960 with a chief complaint of upper abdominal pain of twenty-two years' duration. At first these attacks were episodic and infrequent, characterized by an epigastric burning pain radiating through to the back and associated with prostration.

The patient gave a history of heavy alcoholic intake on week ends and he noted that alcohol would cause exacerbations of the above attacks. He had several admissions without relief. In June, 1959 he was again hospitalized with complaints of a more constant pain. From then until the present admission he was almost constantly a hospitalized patient with daily attacks of pain, unrelieved except by a variety of analgesic and narcotic preparations. He had lost 17 pounds over the previous eight months. Although his pain seemed unrelated to food intake, fatty foods would cause him the most distress. He had no diarrhea, steatorrhea, melena, hematemesis, but had noted definite jaundice.

Physical examination upon admission revealed a chronically ill Negro man in no acute distress with a blood pressure of 114/70 mm. Hg, pulse 100, respiration 18, and temperature 98.4°C. The examination was essentially unrevealing except for the presence of some mild abdominal tenderness in the left upper quadrant.

Studies at another institution just prior to admission showed pulmonary emphysema, intra-abdominal calcification characteristic of pancreatic calcifications, enlargement of the head of the pancreas with an inverted 3 sign and a nonspecific small bowel abnormality. Cholecystography revealed a normal sized gallbladder with good visualization and no stones. A double intensification study visualized a distal obstruction of the common duct with proximal dilatation. Studies revealed liver function to be normal, pancreatic enzymes were low normal. Blood chemistry determinations were within normal limits: cholesterol 159 mg. per cent, creatinine 1.4 mg. per cent, serologic test for syphilis negative, bromsulfalein 2 per cent, routine urinalysis, negative. Examination of feces revealed a trace of occult blood with no increase in fat and presence of undigested meat fibers. A one hundred mc. I^{131} triolein fat meal revealed the following percentages of tagged neutral fat in the blood: two hours 3.3 per cent, four hours 6.4 per cent, 6 hours 8.3 per cent, 8 hours 8.8 per cent, 24 hours 4.5 per cent. These figures represent a low normal appearance of tagged neutral fat in blood.

In view of the clinical history, laboratory and x-ray findings a diagnosis of chronic relapsing pancreatitis was made preoperatively and the patient was explored with the intention of performing a cholecystectomy and sphincterotomy as a primary attack on this problem. At operation, the abdomen was entered through a right subcostal incision. Exploration revealed the common duct enlarged to about 1.5 cm. in diameter and the head of the pancreas was quite firm. Following a routine cholecystectomy, a transverse incision was made in the anterior wall of the second portion of the duodenum. Immediately proximal to the ampulla of Vater, there was a mucosal lesion which on frozen section suggested a polypoid carcinoma of the duodenum. In view of this and no evidence of distant spread, a Whipple type procedure was used, in which the

stump of the pancreas was implanted into the end of the jejunum, the common bile duct implanted into the side of the jejunum, and the stomach stump anastomosed to the side of the jejunum. Postoperatively there were no complications and the patient was discharged on a regular diet on the seventh postoperative day. The final pathologic report was adenocarcinoma arising from a cystadenoma of the pancreas and postinflammatory pancreatic fibrosis. A complete examination of the liver prior to discharge showed no abnormalities.

The patient's condition was followed in the outpatient department and he was found asymptomatic. He was eating well and gaining weight. Four weeks following discharge from the hospital, hematemesis developed. The patient had several recurrent bouts of hematemesis over a twelve hour period and finally presented himself at the Graduate Hospital in a state of impending shock. He had a black stool mixed with bright red blood.

Examination revealed cold, moist skin, blood pressure of 100/58 mm. Hg, pulse 120, temperature 98.6°F. The only finding except for some tenderness deep in the right upper quadrant was the presence of bright red blood on rectal examination. The initial hemoglobin was 8.2 gm. per cent. He received 2 units of blood in the next four hours and a repeat hemoglobin was 8.2 gm. per cent. Another 1,000 cc. of blood brought the hemoglobin to 10.3 gm. per cent. Eight hours following admission, the patient had a bout of massive hematemesis and a large, bright red and tarry stool. He became severely hypotensive with blood pressure 80/30 mm. Hg and pulse 160. The patient received a blood transfusion expedited through use of a blood pump and was immediately taken to the operating room for exploration.

The abdomen was entered through an upper midline incision. The entire gastrointestinal tract showed evidence of blood within its lumen. A gastrotomy was performed in the gastric remnant and visual and digital inspection of the gastrojejunostomy failed to reveal a marginal ulcer or any cause for the bleeding. Bright red blood and clots were noted to be coming from the afferent limb of the gastrojejunostomy. The gastrotomy was closed. An opening was made in the most proximal portion of the jejunum several centimeters distal to its anastomosis with the pancreas. The anastomosis of the jejunum to the pancreas was intact and there was no evidence of any type of ulcer formation in this area. The anastomosis of the common duct with the jejunum was visualized and found patent and normal. Bright red blood was noted to spurt from the common bile duct into the jejunum. The opening in the jejunum was extended up the common duct and the junction of the right and left hepatic ducts was visualized. Bright red blood, in spurts, was noted to be coming from the left hepatic duct.

The incision was extended upwards along the left hepatic duct as far as the liver parenchyma would allow. Bright red blood continued to gush from the left hepatic duct without any visable cause. A suction tip was inserted into the proximal duct to control the bleeding. The hepatic artery was identified, but compression did not influence the flow of blood from the left hepatic duct. A table consultation established that the bleeding was arterial, not apparently controlled by hepatic artery compression. Suggestions were offered as follows: (1) Ligate the left hepatic duct. (2) Introduce a Foley catheter and blow up the balloon. (3) Pack the duct with gauze. (4) Pack the duct with Gelfoam.® During this consultation, the glass tip of the aspirator was inserted into the left hepatic duct to remove the blood as it appeared. After about five minutes of continuous suction, the bleeding seemed to lessen somewhat in amount. It was then decided to try packing the duct with Gelfoam because such a pack would not have to be removed, and it was thought that the Gelfoam would come away of itself after the bleeding was controlled. Long strips of Gelfoam were inserted into the proximal left hepatic duct and packed as tightly as possible. Several large packages of Gelfoam cut in narrow strips were inserted in this manner. The bleeding stopped following this and a No. 18 French rubber catheter was placed in the left hepatic duct just distal to the Gelfoam pack. The catheter was then positioned in the common duct, through the choledochojejunostomy into the jejunum and out the lower portion of the jejunotomy. The left hepatic duct and common duct were closed with a continuous over and over suture of No. 2–0 chromic catgut. The jejunotomy was closed in a similar manner. The catheter and two cigarette drains were brought out through a stab wound. The patient received 5 units of blood during the operative procedure and 5 units prior to surgery. His condition stabilized following the cessation of the hemorrhage. The postoperative course was uncomplicated. The catheter began to drain very dark bile in small amounts immediately postoperatively for forty-eight hours. Over a ten day period, 3,000 cc. of bile were collected. The drainage was clear biliary drainage, free of microscopic blood after the first forty-eight hours after surgery. Stools were grossly acholic until the seventeenth postoperative day when the biliary tube was removed and the stools turned brown. The patient was discharged on the nineteenth postoperative day. Complete studies of liver function prior to discharge revealed no abnormalities. A preoperative elevated transaminase of 330 units had returned to 9 units prior to discharge and there was no evidence of jaundice.

The patient has been closely followed since discharge and it is now eight months since his second operation. He is asymptomatic, free of pain and

eating a regular diet with abstinence from alcohol. He is gaining weight and shows no evidence of any further bleeding or of liver damage.

COMMENTS

Massive upper gastrointestinal hemorrhage of undetermined origin constitutes 2 to 5 per cent of all such bleeding [3]. Of these, about twenty-nine cases of massive hemorrhage originating in the biliary system have been reported [1–5]. Sandblom [4] originally referred to hemorrhage into the biliary tract following trauma as "hemobilia," a term which has now been used in the literature to describe all bleeding into the bile ducts.

Hemobilia may be classified as traumatic or nontraumatic. The traumatic variety usually follows an episode of blunt trauma by several days to weeks and carries a 50 per cent mortality. Fourteen cases have been reported in the literature since Owen first reported it in 1848 [6]. Nontraumatic hemobilia has been reported to result from tumors of the liver or biliary tract; inflammations of the gallbladder or bile ducts as hemorrhagic cholecystitis and biliary calculi; following intubations of the common duct or gallbladder; and from aneurysms of the hepatic artery or cavernous transformation of the portal vein. Stahl [5] has reviewed the literature and reports twelve cases of documented nontraumatic hemobilia in addition to the cases of hemorrhagic cholecystitis he reports. There have been two documented cases of late postoperative hemobilia following surgical manipulation of the common duct. Campbell [2] reported a fatal erosion into the superior pancreaticoduodenal artery after choledochostomy and ulceration of the distal common duct. Andreassen [1] recently reported a case of massive hemobilia following a choledochostomy which was controlled by ligation of the hepatic artery.

The present case report is one of massive upper gastrointestinal hemorrhage occurring one month after a Whipple procedure for a small carcinoma of the head of the pancreas. The site of bleeding was from the left hepatic duct, the exact source was unknown. The bleeding could not be controlled by hepatic artery compression and represents an acute problem in management and a puzzling problem as to cause. It is believed that since the site of hemorrhage was proximal to the left hepatic duct and refractory to hepatic artery occlusion, previous operative trauma was probably not responsible for this hemorrhage and therefore this case represents a spontaneous idiopathic hemobilia.

The management of hemobilia must be individualized for each case, depending on the source of hemorrhage and this may tax the ingenuity of the surgeon. Operative management of hemobilia has been accomplished by a variety of means, including biliary decompression [3]; packing and draining of traumatic cavities [5]; hepatic artery ligation with massive antibiotic support [1]. Bleeding from local disease in the gallbladder and extrahepatic ducts can often be controlled by cholecystectomy and resection or removal of stone, tumor or indwelling tube [5]. In the case presented in this paper, control of bleeding was accomplished by extensive packing of the left hepatic duct with Gelfoam strips.

SUMMARY

1. A case report of idiopathic massive hemobilia is presented.

2. The occurrence, classification and management of hemobilia are discussed.

REFERENCES

1. Andreassen, M. and Lindenberg, J. Late postoperative hemobilia treated with peripheral ligation of the hepatic artery. *Surgery*, 46: 870, 1959.
2. Campbell, D. H. Intracholedochal hemorrhage. *Ann. Surg.*, 141: 125, 1955.
3. Kerr, H. H., Mensh, M. and Gould, E. A. Biliary tract hemorrhage: a source of massive gastrointestinal bleeding. *Ann. Surg.*, 131: 790, 1950.
4. Sandblom, P. Hemorrhage into the biliary tract following trauma: "traumatic hemobilia." *Surgery*, 24: 571, 1948.
5. Stahl, W. M. Jr. Gastrointestinal tract hemorrhage due to gallbladder disease. *New England J. Med.*, 260: 471, 1959.
6. Owen, H. K. Case of lacerated liver. *Lond. M. Gaz.*, 17: 1048, 1848.
7. Manfredi, D. H. Gastrointestinal hemorrhage originating in the biliary tract. *New York J. Med.*, 58: 2397, 1958.

CASE ANALYSIS CLINIC

Pseudomembranous Enterocolitis

THOMAS V. CRAIG, M.D., *Columbus, Ohio*

From the Department of Surgery, Ohio State University Medical Center, Columbus, Ohio.

A FIFTY-TWO year old patient was in excellent health until two weeks prior to admission when she accidently found a moderately tender mass in the right, lower part of the abdomen. There was no change in bowel habits.

PHYSICAL EXAMINATION

Examination showed a seemingly healthy, vigorous woman with a firm, round, freely movable mass measuring 7 cm., deep in the right lower abdominal quadrant.

LABORATORY DATA

Routine blood and urine analyses were normal. X-ray of the chest was normal. A barium enema showed a small polypoid carcinoma of the ascending colon.

EARLY MANAGEMENT AND SURGERY

The patient was prepared for surgery with multiple enemas, Sulfasuxidine® and neomycin. At surgery a right colectomy was performed. An ileotransverse colostomy was constructed without incident.

The first postoperative day passed uneventfully. On the morning of the second postoperative day the temperature suddenly rose to 104°F. The blood pressure dropped to 85/50 mm. Hg with a pulse rate of 150 beats per minute. The abdomen was diffusely tender on palpation. It became distended and the urine output fell off to 60 cc. over a four hour period. By evening the patient had twice passed about 100 cc. of brown liquid stool. Graham stain of a stool smear showed large numbers of gram-positive cocci. The following day the patient passed over 5 L. of bright green, liquid stool containing white flakes of purulent exudate. Stool cultures grew out pure Staphylococcus aureus, coagulase positive. A diagnosis of pseudomembranous enterocolitis was made.

The patient was treated vigorously with saline, blood and plasma. Erythromycin was given in doses of 1 gm. four times a day to arrest the growth of the staphylococci. Twenty-five units of ACTH were administered four times a day to counteract the systemic effect of the staphylococcal enterotoxin. The stool output remained at 5 L. for an additional day, then rapidly fell to normal over the next three days with marked improvement in the general status of the patient. The temperature returned to normal. By the seventh postoperative day the stool was free of staphylococci and the patient was discharged symptom free ten days following operation.

COMMENTS

Pseudomembranous enterocolitis varies in severity from the rapidly fatal cases on the one hand to harmless lingering diarrheas on the other. If pseudomembranous enterocolitis occurs in the postoperative period, there is a very poor prognosis for survival. The mortality rate in most series approaches 70 per cent in spite of modern supportive measures.

The clinical appearance in this disease can be baffling and the diagnosis difficult. Shock and death can occur with incredible rapidity in an otherwise normal postoperative course. The etiology remains confused. Although enterocolitis was seen and treated before the era of antibiotics, its frequency has apparently increased with the advent of broad spectrum antibiotics. All antibiotics have been incriminated but there is no constant relationship between their

administration and the disease. The disease is most frequently seen in patients subjected to operation but is also seen in medical patients.

The etiology can be broadly classified into two groups: (1) cases occurring after the administration of an antibiotic, the "diarrheal type"; and (2) cases in which no etiologic agent is apparent, the "collapse type." The usual idea regarding the first type is that enterocolitis ensues after suppression or alteration of the normal gastrointestinal flora by antibiotics. This permits the staphylococcus to achieve pathologic proportions. The experiments of Prohaska [1] lend support to this theory. He has shown that the staphylococcus cannot flourish in the normal gastrointestinal tract.

The diarrheal type of the disease is associated with use of antibiotics but it is not necessarily preceded by surgery. At the onset in this type there is fever with progressive abdominal distention and tenderness. The distention is ascribed to a paralytic ileus; however, there are no x-ray findings peculiar to this condition. Profuse watery diarrhea of bright green color with large white flakes of purulent exudate is characteristic and occurs within a few hours. The Staph. aureus can invariably be grown out in pure culture. Fluid loss may range from moderate to tremendous. In severe cases a fluid exchange of 8 to 12 L. per day is possible. This is twice the normal blood volume, which points up the necessity for accurate fluid and electrolyte replacement. Shock ensues rapidly and is due to both hypovolemia from fluid loss and widespread vasodilatation caused by the staphylococcus enterotoxin.

The second group consists of cases of pseudomembranous enterocolitis not associated with antibiotics or infection by the staphylococcus. This is the "circulatory collapse" type described by Hardaway and McKay [2]. These authors describe an intravascular coagulation of the capillaries of the submucosa of the bowel. They do not clearly explain the mechanism of intravascular clotting, however. They have reproduced this condition experimentally by injecting incompatible blood into the mesenteric vessels of dogs. On the other hand, Hale and Cosgriff [3] have reported full thickness necrosis of the bowel wall in the absence of intravascular clotting.

This type usually occurs in postoperative patients. There is widespread ischemic necrosis and slough of the bowel mucosa. It is characterized by a precipitous drop in the blood pressure. The blood pressure and pulse may be unattainable yet the patient's body may be warm to the finger tips. Fever varies from 101° to 107°F. Diarrhea may or may not be present. The abdomen becomes distended and excruciatingly tender. The patient may vomit dark blood and pass dark, bloody stools containing casts of pseudomembrane. There may be hematuria. The shock observed here is refractory to all but heroic measures. Cooper and Ferguson [4] believe adrenal failure exists in some instances, as shown by a high total eosinophil count. The patient's general condition improves dramatically with cortisone therapy, along with a drop to normal in the eosinophil count.

TREATMENT

There are three phases of treatment: The first and most important is replacement of fluid and electrolytes. An accurate intake output record should be maintained. Urine volume and specific gravity are recorded every two to three hours. These studies show both the degree of dehydration and the effectiveness of fluid replacement. Serum, urine, and stool electrolyte content should be measured frequently to facilitate accurate replacement. The stool electrolyte content may be anticipated to be about as follows: sodium, 70 to 90 mEq./L.; potassium, 10 to 20 mEq./L.; chloride, 90 to 100 mEq./L. Shock must be combated with blood and plasma. There is further rationale for the use of plasma in this disease, since there is considerable inflammatory exudate. During the first twenty-four hours, 500 cc. of whole blood and 500 cc. of plasma will generally be required.

The second phase consists of discontinuing all antibiotics being given at the time. Their action has presumably created a favorable environment for overgrowth of the Staph. aureus. Stool cultures and sensitivities should be made and the appropriate antibiotic given. If Erythromycin or Chloromycetin® have not been in use, then they should be employed in treatment. These two drugs, which have great effectiveness against the Staph. aureus, should be employed in doses of 1 gm. four times a day.

The third phase of treatment involves the use of ACTH and cortisone. Prohaska et al. [5] have reported dramatic results in treating patients with severe pseudomembranous entero-

colitis with 25 units of ACTH four times a day. They maintain that ACTH has a specific antagonistic effect against the staphylococcus enterotoxin. We used ACTH in the case presented above with rapid resolution of the disease process. The recommended dosage is 25 units four times a day.

Cortisone does not seem to be as effective as ACTH except if sudden shock or other signs of adrenal insufficiency occur. In the latter instances it should be used in doses of 200 mg. by intravenous drip twice a day.

As the patient improves, the dosages of ACTH and cortisone should be reduced at a rapid rate. Antibiotics are discontinued when repeated stool cultures fail to grow out staphylococcus.

An adjunct to be considered when hypotension occurs, is norepinephrine. Staphylococcus enterotoxin causes marked peripheral vasodilatation which can be reversed by norepinephrine. Lastly, in intractable cases where the Staph. aureus is present in the stool, fecal enemas from normal donors should be tried. Eiseman et al. [6] have had very good results by giving a saline suspension of normal feces as a retention enema in cases where Erythromycin and cortisone were ineffective. In three of four cases, this enema was given on two successive days. In each case improvement occurred within twenty-four hours of the last enema.

REFERENCES

1. Prohaska, J. V., Long, E. T. and Nelson, T. S. Pseudomembranous enterocolitis. Its etiology and mechanism of the disease process. *Arch. Surg.*, 72: 977, 1956.
2. Hardaway, R. M. and McKay, D. G. Pseudomembranous entercolitis. *Arch. Surg.*, 78: 446, 1959.
3. Hale, H. W. and Cosgriff, J. H. Pseudomembranous enterocolitis. *Am. J. Surg.*, 94: 710, 1957.
4. Cooper, D. R. and Ferguson, L. K. Acute enterocolitis. *Am. Surgeon*, 23: 165, 1957.
5. Prohaska, J. V., Govostes, M. C. and Taubenhaus, M. Postoperative pseudomembranous enterocolitis. Successful treatment with ACTH. *J. A. M. A.*, 154: 320, 1954.
6. Eiseman, B., Silen, W., Bascom, G. S. and Kauvar, A. J. Fecal enema as an adjunct in the treatment of pseudomembranous enterocolitis. *Surgery*, 44: 854, 1958.

PRACTICAL SURGICAL SUGGESTION

An Investigation of Plastic Dressings for Clinical Use

JOSEPH M. MILLER, M.D.,* DEBORAH A. JACKSON, B.S. AND CHARLES S. COLLIER,
Baltimore, Maryland

From the Fort Howard Division, Veterans Administration Hospital, Baltimore, Maryland.

PLASTIC dressings for clinical use usually consist of a suitable ingredient dissolved in an organic solvent to make a solution capable of forming a film following the evaporation of the solvent. The substances in current use include vinyl resin plastics, acrylic resins and cellulose acetates. In some instances a softening agent or a plasticizer is added to the solution to make the film soft and pliable. This permits the film to conform readily to the surface to which the film is applied. One or a combination of two of the fluorinated hydrocarbons are used as the propellant.

Aeroplast is a solution of the co-polymers of hydroxy-vinyl chloride-acetate and sebacic acid, 9.3 per cent by weight, and a modified maleic rosin ester, 3.1 per cent by weight, in ethyl acetate and acetone. A fluorochlorohydrocarbon gas is the propellant.

Rezifilm® base is a solution of a methacrylate resin in ethyl acetate. In Rezifilm, tetramethylthiuram disulfide is added to the solution as an antimicrobial agent. The propellants are dichlorodifluoromethane and trichloromonofluoromethane.

Scan® spray-on dressing S-1 is a solution of cellulose acetate butyrate plasticized with 2-4 ethylhexyl diphenyl phosphate in acetone. In Scan spray-on dressing S-2, dequadinium chloride (decamethylene-bis-[4-amino quinaldinium chloride]) was added as an antimicrobial agent. The propellant is a mixture of dichlorodifluoromethane and trichloromonofluoromethane.

In general the physical properties of these dressings are similar. They adhere well to the skin and are neither irritating nor sensitizing. They provide a transparent dressing for a wound, are tough and flexible and remain in place despite friction or stress of motion.

These dressings have much to recommend their use clinically. The related problems of antimicrobial action and permeability are important if their use is intended to prevent bacteria from entering a clean wound. Studies of these problems were made, therefore, using the three different types of dressings.

ANTIMICROBIAL ACTION

Blood agar plates were streaked with broth cultures of the various bacteria, and Sabouraud agar plates were inoculated with the fungus. The plates were then thoroughly sprayed with one of the indicated substances and were incubated at 37°C. for twenty-four hours. The results are shown in Table 1.

The same substances were used to make a film on sterile water. The membrane was left in place for two minutes. The film was then removed and allowed to dry in an incubator at 37°C. for ten minutes. Corresponding plates were inoculated in a similar manner with the same microorganisms. Small pieces of the dried films were placed upon the agar. The preparations were incubated at 37°C. for twenty-four hours. The results were the same as shown in Table 1.

Sterile discs of filter paper were sprayed with the substances on one side and left to dry for three hours. Corresponding preparations of the

* Department of Surgery, School of Medicine, The Johns Hopkins University, Baltimore, Maryland.

TABLE 1

ANTIMICROBIAL ACTION OF THE INDICATED DRESSINGS ON VARIOUS BACTERIA

Substance	Microorganisms						
	Staphylococcus Aureus	Streptococcus Pyogenes	Escherichia Coli	Proteus Vulgaris	Pseudomonas Aeruginosa	Aerobacter Aerogenes	Candida Albicans
Control	+*	+	+	+	+	+	+
Aeroplast	+	+	+	+	+	+	+
Rezifilm base	+	+	+	+	+	+	+
Rezifilm	−†	−	+	+	+	+	−
Scan spray-on dressing S-1	+	+	+	+	+	+	+
Scan spray-on dressing S-2	−	−	+	+	+	+	−

* Growth present.
† Growth not present.

microorganisms were made. The discs were put on the plates with the sprayed side in contact with the agar. The plates were incubated at 37°C. for twenty-four hours. The results were the same as shown in Table 1.

PERMEABILITY OF FILM

Penetration of the film by water and bacteria is an important consideration when attempts are made to loculate a clean wound from external contamination. The amount of moisture on a surface, however, will determine the type of film made. On a dry surface it should be possible to make an impermeable membrane. When the surface is wet, the molecules of the plastic dressing should not align themselves in a coordinated way to achieve impermeability. In addition the more times a given area is sprayed with one of these substances, the greater are the chances that an impermeable membrane will be formed; likewise, the smaller the area sprayed, the greater will be the chance of an impermeable membrane being formed. From a practical standpoint, two sprays to an area were applied to determine permeability of the films.

Blood agar plates were inoculated with a broth culture of the Staphylococcus aureus. The individual preparations were then sprayed twice with aeroplast, Rezifilm base or Scan spray-on dressing S-1. The plates were incubated at 37°C. for twenty-four hours. Growth of the bacteria was present under the film on all plates. In the center of each plate was placed 0.5 ml. of broth. The fluid passed through the film very rapidly. After three hours, 0.5 ml. of broth was again placed on each plate. A subculture of this fluid was made to blood agar plates and these were incubated under similar conditions. The Staph. aureus was recovered in each instance, thus demonstrating the passage of the bacteria through the film.

Under sterile conditions, cylinders of metal measuring 6 mm. in diameter were placed in agar. The surface of the agar was sprayed twice with one of the substances being tested. The film was allowed to dry for one hour. In each cup was placed 0.1 ml. of a broth culture of Staph. aureus. The preparations were incubated at 37°C. for twenty-four hours. The same quantity of broth was then added to each cup. After five hours subcultures to blood agar plates were made and incubated. The original preparations were incubated for an additional twenty-four hours. The Staph. aureus was isolated from the cups of each preparation. The cups were then carefully removed from the agar. Subcultures of the agar from the center of the spot from which the cups were removed were made into broth. The Staph. aureus was recovered from all preparations. This data indicated that the Staph. aureus could pass through each of the films.

Experiments using a film on a dry surface were then made. Serratia marcescens was employed as the test bacteria.

An aliquot of a broth culture of the microorganism was put on a sterile slide and this was

allowed to dry in an incubator at 37°c. for one hour. The preparation was then placed in broth and incubated at room temperature for twenty-four hours. The bacteria were recovered from the broth.

Blood agar plates were then streaked with the microorganism and these were sprayed twice with either aeroplast, Rezifilm base or Scan spray-on dressing S-1. The preparations were incubated at room temperature for twenty-four hours. The bacteria were not affected by any of the dressings.

Having shown that the Serratia marcescens was not killed when put on glass slides and that the dressings were not bactericidal for it, preparations of the bacteria on glass slides were made and allowed to dry as was done previously. The slides were sprayed twice with one of the dressings and dried in an incubator at 37°c. for one hour. The glass slides were then put in broth at room temperature for twenty-four hours. The S. marcescens was not recovered from any of the preparations.

An *in vivo* test using the same microorganism on the skin of patients was then made since this experiment would approach the conditions of clinical use. The S. marcescens in a broth culture was placed on a small area of the skin of the abdomen of patients. The sites were then sprayed twice with either aeroplast, Rezifilm base or Scan spray-on dressing S-1. There were six patients in each group. The patients were requested to abstain from washing these areas during the test period of twenty-four hours, although they were not prohibited from other activity in the normal routine of the hospital. Cultures were then taken from the film and inoculated in broth and incubated at room temperature for twenty-four hours. Transfers to blood agar plates were made with incubation under similar conditions. The S. marcescens was not recovered from any of the films.

COMMENTS

These investigations demonstrated certain facts about the plastic dressings used. Aeroplast does not have an antimicrobial effect against any of the microorganisms tested while Rezifilm and Scan spray-on dressing S-2 produced such action only on some organisms. The tetramethylthiuram disulfide in the Rezifilm and the dequadinium chloride in the Scan spray-on dressing S-2 diffused from their film and from the filter paper wet with these dressings to produce the same microbicidal action. None of these dressings produced a film impermeable to water and Staph. aureus when the films were made on a wet surface. The situation was changed when a dry surface was used, for these films were impermeable to the S. marcescens.

Certain statements about the clinical use of these dressings may be made in the light of this investigation. All areas should be sprayed at least twice to avoid bare spots. An impermeable film will only be obtained on a dry surface. The degree of impermeability apparently is directly related to the amount of water on the surface receiving the film. In slightly wet areas the film will keep gross contamination from the selected area but bacteria will pass through the membrane. It is wise to add an antimicrobial substance to the dressing, therefore, to kill the bacteria passing through a permeable film. If such a chemical is used, it should be able to diffuse easily from the film. The antibacterial substances used in the present study are not effective against certain microorganisms which can cause disease.

SUMMARY

Three plastic dressings for clinical use were investigated. The surface on which these dressings are used must be dry to obtain an impermeable film. An antimicrobial substance is a necessary additive to such dressings.

A Hand Stapler for Use in Radical Mastectomy

HERBERT VOLK, M.D., *New York, New York*

From the Department of Surgery, Albert Einstein College of Medicine of Yeshiva University and Bronx Municipal Hospital Center, New York, New York.

RADICAL mastectomy, one of the most frequently performed surgical procedures for cancer, should be carried out with especially gentle technic. Undue manipulation of the cancerous breast can initiate embolization of malignant cells. Thus, the surgeon should utilize as many aids as practical to prevent iatrogenic dissemination.

It has been the custom at this center, as well as others, to attach a surgeon's sterile cap to the skin edges of the operative specimen. Traction is then exerted on the cap to provide exposure and avoid pressure on the underlying breast.

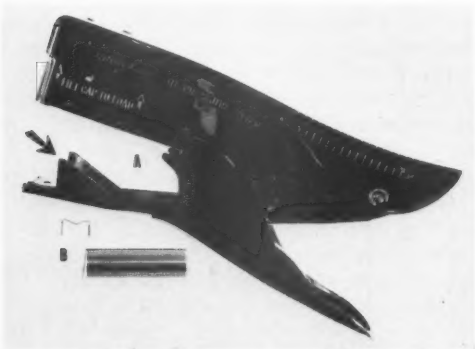


FIG. 1. Hand stapler. A, arrow points to guard which assures staples will be inserted close to edge of specimen. B, staples are standard 5/16 inch size.

This communication describes a hand stapler which has facilitated attaching the cap to the edges of the skin.

Previously, the surgeon's cap was attached to the breast specimen by fine hemostats which were then tied together in groups. This arrangement was unsatisfactory; the clamps were bulky and interfered with exposure.

Michel clips were tried; this eliminated the bulk factor, but applying the clips was time consuming and many pulled loose as traction was applied to the cap.

It seemed that a hand stapler might be feasible for this purpose. Initially, a stock commercial model was used. This appliance worked well but did not tolerate repeated autoclaving. With the cooperation of the manufacturer,* the same model was constructed of stainless steel and has been in use for over three years and in fifty operations. The only modification of the commercal apparatus was the addition of the vertical guard ½-inch from the stapling edge. (Fig. 1A.) This guard assures that the staples will be inserted close to the edge of the incision. Regular stock staples are used (Fig. 1B.)

Preoperatively, the stapler containing a cartridge of staples is autoclaved. A spare cartridge is also autoclaved, although one length usually suffices for the entire circumference of the operative specimen. The staples can be autoclaved only once as a second autoclaving causes the staples to separate due to the destruction of the glue which binds the strip.

The use of this stapler shortens operating

* Swingline, Inc. (Hi-Pressure Plier).

FIG. 2. Surgeon's sterile cap being stapled to edge of specimen. Note the vertical guard ½ inch behind stapling head.

time, since the cap can be attached to the edges of the skin rapidly and with facility. In addition, the staples provide hemostasis on the specimen side as is illustrated in Figure 2. Thus, clamping and ligating or coagulating specimen skin bleeders are eliminated.

CONCLUSION

A stainless steel hand stapler, which is simple to use, has been described. This apparatus facilitates the attachment of a surgeon's sterile cap to the radical mastectomy specimen.

The Use of Intramuscular Perphenazine to Control Postoperative Vomiting

A REPORT OF 2,794 CASES

BERTON E. BRALY, M.D. AND DANIEL C. MOORE, M.D., *Seattle, Washington*

From the Department of Anesthesia, The Mason Clinic, Seattle, Washington.

A PREVIOUS double blind study to determine the effectiveness of perphenazine (Trilafon®) in controlling postoperative vomiting has been reported [12]. It showed that incidence of vomiting postoperatively in the group of patients receiving perphenazine was 6.25 per cent (304 patients, nineteen vomited) while in those patients who received the solvent only, the incidence of vomiting was 17.97 per cent (306 patients, fifty-five vomited). In this survey of cases in which perphenazine was employed, the P value was less than 0.001. This indicated a highly significant reduction in the rate of postoperative vomiting when this drug was used; that is, less than one time in a thousand could such a reduction be attributed to chance if the perphenazine were not effective. During this double blind study, there were no side effects from the dosage of perphenazine administered (3.75 mg.).

While there are numerous reports on the effectiveness of perphenazine in controlling vomiting associated with disease, studies on its effectiveness to control postoperative vomiting are few and have been limited in the number of patients studied [1–3,5–7,12,13,16–18,22]. Because of this and our belief that a larger series (2,000 or more cases) using a solution known to contain a specific dose of perphenazine would either substantiate or contradict the double blind study as well as reveal some of the complications associated with the administration of the drug, perphenazine was administered to 2,794 patients who were to undergo surgical procedures requiring anesthesia.

METHOD OF STUDY

The general method used by us in previous studies of antiemetic drugs was employed [9]. For perphenazine, it consisted of the intramuscular injection of ¾ cc. (3.75 mg.) of perphenazine on going to surgery, again on return from surgery and every six hours thereafter for a total of four doses. All surgical patients who were to receive an anesthetic were included, except children under twelve years of age, regardless of the type of surgery, anesthesia or premedication to be used. Any patient who happened to receive another specific antiemetic, e.g., dimenhydrinate (Dramamine®), was excluded from the report. Premedication consisted of either atropine sulfate or scopolamine hydrobromide plus an opiate such as morphine sulfate or meperidine (Demerol®). Many of the patients who were to have regional block procedures also received a barbiturate.

The study was restricted to vomiting because of the difficulty in evaluating the psychic factors involved in the subjective symptom of nausea. Vomiting was defined as emesis of any material which measured 50 cc. or more in

TABLE 1
INCIDENCE OF VOMITING IN PATIENTS RECEIVING PERPHENAZINE

Type of Anesthesia	Total No. of Cases	No. of Patients Who Vomited	Percentage Who Vomited
Ether + nitrous oxide	288	37	12.9
Cyclopropane	113	7	6.2
Thiopental + nitrous oxide + muscle relaxant	610	49	8.0
Thiopental sodium	25	1	4.0
Regional nerve block*	490	16	3.3
Spinal	606	16	2.6
Epidural			
Caudal	166	4	2.4
Lumbar area	477	15	3.1
Dolitrone®	19	0	0
Total	2,794	145	5.2

* Block of nerves after they have emerged from the intervertebral foramina.

volume. Spitting up of mucus or bile was not considered vomiting in this study. All patients in these statistics were seen before operation, within the first twenty-four hours after operation and daily thereafter. They were questioned as to whether or not they had vomited, and asked to estimate the amount or volume involved. Their responses were checked with the notes taken by the nurses. These findings, as well as unusual side reactions when they occurred, were noted on a "keysort" type of record.

RESULTS

Antiemetic Effect. A total of 2,794 cases received perphenazine. Of that number, 145 or 5.2 per cent of the patients vomited during the time they were receiving the perphenazine. (Table 1.) There was no correlation between the frequency of emesis and the amount vomited. Those who did vomit quite often, vomited large amounts of from 200 to 400 cc. or more.

Complications. Hypotension in the immediate postoperative period (one to six hours) seemed to occur more frequently during this study than it is normally encountered, but this was strictly a clinical impression. Prolonged recovery time in a few cases seemed to be attributable to the combination of the anesthetic agent and the perphenazine.

There were four cases of dystonia characterized by spasms of the muscles of the neck progressing to torticollis, extensor rigidity of the muscles of the back, carpopedal spasm, trismus and difficult swallowing. In one of the four cases, the muscle spasm of the diaphragm and intercostal muscles was so marked that the patient became cyanotic and required oxygen therapy. There was no evidence of local reaction or pain at the site of injection.

COMMENTS

With the advent of modern anesthesia, postoperative vomiting has been lessened but continues to be a problem to the anesthetist, the surgeon and the patient. Numerous drugs with antiemetic activity have been introduced and applied to this problem in the past few years. A method was developed at our institution in 1951 to study the clinical effectiveness of some of these drugs [9]. The study reported herein represents a continuation of these trials.

The over-all incidence of vomiting for the various anesthetic technics used at our institution when no antiemetic drug was employed has been established by five studies of separate series of patients with a total of 2,283 patients. The results are as follows: Study I, 1,020 patients, 217 vomited (21.27 per cent); Study II, 482 patients, 122 vomited (25.3 per cent); Study III, 199 patients, forty-eight vomited (24.1 per cent); Study IV, 276 patients, seventy-seven vomited (27.9 per cent); and Study V, 306 patients, fifty-five vomited (17.97 per cent) [9–12]. Studies III, IV and V were the controls in double blind studies and during their execution neither the members of the Department of Anesthesia, the floor nurses nor the surgeons knew which patients received the solvent alone and which received the solvent containing the antiemetic drug. An average of these control studies indicates that the incidence of vomiting when no antiemetic drugs were used is approximately 22.7 per cent in our institution. (Table II.)

The incidence of vomiting after administration of perphenazine in this study of 2,794 cases was 5.2 per cent. This compares favorably with the incidence of vomiting in the series with perphenazine of the previously conducted double blind study or 6.2 per cent [12]. Of the antiemetic drugs which had been subjected to

TABLE II
CONTROL SERIES

Type of Anesthesia	Total No. of Cases	No. of Patients Who Vomited	Percentage Who Vomited
Ether + nitrous oxide	426	147	34.5
Cyclopropane	160	60	37.5
Thiopental + nitrous oxide + muscle relaxant	284	61	21.5
Thiopental sodium	278	48	17.3
Regional nerve block*	432	58	13.4
Spinal	453	97	21.4
Epidural			
Caudal	94	19	20.2
Lumbar area	156	29	18.6
Total	2,283	519	22.7

* Block of nerves after they have emerged from the intervertebral foramina.

the previously described clinical method of testing, perphenazine was the most effective in controlling postoperative vomiting. Dimenhydrinate reduced the incidence of vomiting from approximately 24.1 per cent to approximately 13.3 per cent [*10*]. Cyclizine lactate (Marezine®) reduced the incidence of vomiting from approximately 27.9 per cent to approximately 15 per cent [*11*]. Although the antiemetic effects of chlorpromazine (Thorazine®) and prochlorperazine (Compazine®) have not been studied by us, reports indicate that, experimentally and clinically, perphenazine exceeds both in antiemetic potency for equal doses [*15,21*].

The side effects seen with perphenazine (i.e., hypotension and dystonia) were not observed when dimenhydrinate and cyclizine lactate were studied. Some of the surgeons and members of the house staff believe strongly that there was an increased incidence of postoperative hypotension in the patients who received perphenazine. Many of these instances of hypotension were checked by anesthesiologists on the staff and were thought to be due in part to depression from anesthetic drugs or opiates used to control postoperative pain. Postoperative medication for pain was given in the usual dosage during this study, but it is likely that reduction in dosage could be made because of the possible potentiating effect of perphenazine. No difficulty in maintaining blood pressure was encountered during surgery. In the nonsurgical patient, perphenazine produces no change in the respiration, pulse, cardiac rate, blood count or blood pressure [*19,20*]. Nevertheless, because of possible potentiation of barbiturates, opiates and other depressant drugs (anesthetics) by the phenothiazine compounds, perphenazine in combination with these drugs may have been responsible for the observed postoperative hypotensions.

The most dramatic of the complications observed following administration of perphenazine was dystonia. The onset was quite sudden. A primary characteristic of the symptom was its intermittency. It lasted several minutes, disappeared and then occurred again. There was no loss of consciousness and definite prodromas were usually present; for example, the patient anticipated the onset of symptoms and exhibited some excitement when they appeared. Initially, these intermittent symptoms occurred in a crescendo of intensity. Then, as the effect of the drug wore off the intervals between the occurrence of symptoms increased and the intensity of the symptoms subsided. The demonstrations of dystonia which followed the administration of perphenazine have been reported recently by others [*8,14*].

Despite the similarity to symptoms of neurologic disorders such as convulsions, the dystonia subsided gradually within a few hours, and almost always within twelve to twenty-four hours, after the administration of the drug was discontinued. No untoward residual effect from dystonia occurred.

Treatment of dystonia was symptomatic and conservative. Reassuring the patient was sufficient therapy in three of the cases; in the fourth case oxygen therapy in conjunction with reassurance sufficed. Although the administration of sedation or an antiparkinsonism agent such as trihexyphenidyl (Artane®), cycrimine hydrochloride (Pagitane®), benztropine (Cogentin®) or procyclidine (Kemadrin®) has been reported to produce a rapid and dramatic reversal of symptoms in adults with moderate to severe dystonia, we have not used these drugs [*4*].

CONCLUSIONS

Perphenazine can be used routinely in surgical patients as an extraordinarily effective antiemetic. Administration should be with care because of the possibility of side effects such as

hypotension, extrapyramidal stimulation and prolonged anesthesia recovery time. These side effects are infrequent, and the greatest danger in the use of perphenazine routinely may lie in its remarkable antiemetic potency. It is conceivable that the detection of serious postoperative complications such as intestinal obstruction, increased intracranial pressure, gastric dilatation and others might be obscured by suppression of emesis.

SUMMARY

1. Perphenazine has been used for the control of postoperative vomiting in 2,794 cases. The incidence of vomiting was 5.2 per cent in these 2,794 patients, compared with 22.7 per cent in 2,283 patients or the control group.

2. Side effects included hypotension and dystonia.

3. A warning is given regarding the possibility of masking organic surgical complications because of the remarkable antiemetic potency of perphenazine.

REFERENCES

1. Albert, C. A., Therganokar, R. D., Rafii, A., Henley, E. E. and Albert, S. N. The place of perphenazine (Trilafon) in anesthesia, in press.
2. Anderson, G., Albert C., Henley, E., Witten, S., Winschel, A. and Albert S. The effect of Trilafon on nausea and vomiting during labor. *Obst. & Gynec.*, 13: 504, 1959.
3. Cannistra, F. and Abrams, A. S. Perphenazine in obstetrics. Evaluation of its use to replace an analgesic. Unpublished material.
4. Compazine: W.A.M.:C[illegible]. Philadelphia, 1959. Smith, Kline and French Laboratories.
5. Dobkin, A. B. Perphenazine in Clinical Anesthesia. In press.
6. Ernst, E. M. and Snyder, A. M. Trilafon in nausea and vomiting, and anxiety states. *Pennsylvania M. J.*, in press.
7. Gready, T. G., Jr., Estrada, W. J. and Haden, J. Intramuscular perphenazine in labor: study of 700 patients. *Obst. & Gynec.*, 77: 412, 1959.
8. Montgomery, R. D. and Sutherland, V. L. Acute dystonia reaction to perphenazine. *Brit. M. J.*, 1: 215, 1959.
9. Moore, D. C., Anderson, L., Wheeler, G. and Scheidt, J. The use of parenteral Dramamine to control postoperative vomiting: report of 1,192 cases. *Anestbesiology*, 13: 354, 1952.
10. Moore, D. C., Bridenbaugh, L. D., Green, J. C., Piccioni, V. F., Adams, P. A. and Lindstrom, C. A. Intramuscular use of dimenhydrinate (Dramamine) to control postoperative vomiting. *J. A. M. A.*, 159: 1342, 1955.
11. Moore, D. C., Bridenbaugh, L. D., Piccioni, V. F., Adams, P. A. and Lindstrom, C. A. Control of postoperative vomiting with Marezine: double blind study. *Anestbesiology*, 17: 960, 1956.
12. Moore, D. C., Bridenbaugh, L. D., Van Ackeren, E. G. and Cole, F. V. Control of postoperative vomiting with perphenazine (Trilafon): double blind study. *Anestbesiology*, 19: 72, 1958.
13. Preisig, R. and Landman, M. E. The use of Trilafon (perphenazine) in anxiety states and hyperemesis: report of 135 cases. *Am. Pract. & Digest Treat.*, in press.
14. Robbie, D. S. Post-anaesthetic vomiting and antiemetic drugs. *Anaestbesia*, 14: 439, 1959.
15. Rosenkilde, H. and Govier, W. M. A comparison of some phenothiazine derivatives in inhibiting apomorphine-induced emesis. *J. Pbarmacol. & Exper. Tberap.*, 120: 375, 1957.
16. Scurr, C. F. and Robbie, D. S. Trials of perphenazine in the prevention of postoperative vomiting. *Brit. M. J.*, 2: 922, 1958.
17. Sprogis, G. R., Babcock, S. D. and Segall, J. Treatment of psychiatric disorders in mentally deficient patients with Trilafon (perphenazine). *J. M. Soc. New Jersey*, in press.
18. Stewart, B. L. Perphenazine (Trilafon) in urology and surgery. *Am. Pract. & Digest Treat.*, 10: 845, 1959.
19. Trilafon: TR-20, pp. 3. Bloomfield, 1957. Schering Corp.
20. Trilafon: TR-138, pp. 3. Bloomfield, 1958. Schering Corp.
21. Wang, S. C., Amols, W. and Merritt, H. H. Neural mechanisms of emesis and antiemesis. Scientific Exhibit presented at the 106th Annual Meeting, American Medical Association, New York, 1957.
22. Weiss, S., Weiss, J. and Weiss, B. Symptomatic relief of nausea and vomiting with Trilafon (perphenazine): preliminary report. *Am. J. Gastroenterol.*, 29: 173, 1958.

Our Surgical Heritage

D. P. HALL, M.D., *Surgical Department, University of Louisville, Louisville, Kentucky*

CANADA AND UNITED STATES OF AMERICA

JAMES DOUGLAS

JAMES DOUGLAS was born in Scotland in 1800. He began his surgical training as an apprentice to a "surgeon and apothecary." He then journeyed to Edinburgh where he enrolled as a pupil of Dr. Robert Liston, one of the most famous Scottish surgeons of that era. At the age of eighteen years he signed on as surgeon to a whaler, on a trip to the Arctic Circle. He became a licentiate of the Royal College of Surgeons of Edinburgh. Douglas took residence in London and became a student of surgery at Guy's Hospital with Sir Astley Cooper and at St. Bartholomew's Hospital under the great Abernethy.

Following his sojourn in London he again signed on as a ship's surgeon and made a trip to India. His next adventure was that of joining a party of English settlers bent on colonizing the Mosquito Coast. Douglas became sick and was removed in an unconscious condition to a ship and transported

to Boston. After his recovery he became a resident of Utica, New York, where he married and began the practice of medicine in earnest. The Berkshire Medical Institute, an affiliate of Williams College, New York, granted him an honorary Doctor of Medicine degree in 1825. Douglas was implicated in an unfortunate body snatching incident, and thereafter moved to Quebec where he began to practice medicine. While building up a practice he taught anatomy, originally in his home and later in a building granted by the government for this work. In 1831 Douglas started an excellent school of practical surgery at the Marine and Emigrant Hospital where surgical patients were numerous. It is said that before the use of anesthesia he removed the lower jaw of a hypnotized patient without causing any pain.

Asiatic cholera swept Europe during 1832. The Inspector of the Military Hospitals in Canada claimed that it could not cross the Atlantic Ocean. Douglas publicly questioned this man and publicized the fact that the disease would strike Canada during the summer of 1833. He was correct in his prognostication. Douglas rented a house at Beauport in 1847, for the reception of private patients with typhus fever. Douglas can be described as having a deep voice, a powerful physique and a domineering personality. It would seem that it was very easy for him to earn large sums of money but just as easy for him to lose it in bad investments.

He lived with a son at Phoenixville, Pennsylvania, the last eleven years of his life and died at the age of eighty-six years in 1886. His was the pioneer fiber that developed practical surgery in Canada and the northern part of the United States in that period.

"No farther seek his merits to disclose, or draw his frailties from their dread abode."

ROBERT LISTON

Robert Liston was born at Ecclesnachen in Linlitheow, Scotland. At the age of sixteen years, Liston was a pupil of John Barclay in Edinburgh, who was a well known anatomist of the time. He became a student of surgery at Edinburgh Royal Infirmary, proceeding to the London Hospital and St. Bartholomew's Hospital. In 1818 he qualified as a member of both the London and Edinburgh Colleges of Surgeons. After a short time as demonstrator for his old teacher Barclay, he with his cousin, James Syme, founded a school of Anatomy and Surgery (1819). The proprietors of this new school were vexed by the difficulty of securing bodies for dissection in Edinburgh and the school lasted only five years, although very successful.

Liston became a surgical clerk at the Royal Infirmary in Edinburgh, attached to the wards in the outpatient department. He had no regular appointment at the Royal Infirmary and performed most of his surgery in the homes of patients, with his cousin Syme as assistant. His popularity as a surgeon became immense and unfortunately a vile campaign was directed against him; a letter soon followed from the administrator of the Royal Infirmary dismissing him. For five years his surgical career was at a low ebb, not having an infirmary appointment, and it was during this time that he invented and advocated the long splint which was used for so many years.

In 1834 Liston was appointed Professor of Surgery at the University College and surgeon to the University College Hospital in London. It was here that he received his greatest acclaim and became one of the best known surgeons in Great Britain.

The first major operation in England using ether anesthesia was performed at the University College Hospital by Robert Liston in 1846, the operation being a mid-thigh amputation. He was a man of great physical strength and his arms and hands have been likened to those of Hercules. It has been said that he would amputate the thigh single-handed by com-

pressing the artery with the left hand, using no tourniquet, and doing all the cutting and sawing with the right hand.

He will probably be remembered for his "Elements of Surgery" published in 1830 and a "Treatise on Practical and Operative Surgery" published in 1837.

He died at the age of fifty-three years on December 17, 1847, from an aneurysm of the aorta. It was truly said of him that he "was a teacher more by what he did than by what he said." "He was the bearer of a good report, which proved his good taste."

Surgery Court Cases

HOWARD NEWCOMB MORSE,* *Chicago, Illinois*

TILLER VS. VON POHLE

Supreme Court of Arizona

72 Ariz. 11, 230 P. 2d 213

The patient was in ill health and consulted a surgeon, who diagnosed her condition as being extra-uterine pregnancy and who advised her that an immediate abdominal operation was necessary. She submitted to such operation, which was performed by the surgeon. Immediately following the operation severe pains developed in the abdominal region at about the same place where he had made the incision, and for two years thereafter the patient suffered extreme and excruciating pain in that part of her body.

Following the operation the patient, on several occasions, consulted the surgeon and complained to him of her painful condition; on these occasions he assured her that the operation had been a complete success and that her suffering was caused by common gas pains. At other times following the operation, he diagnosed her malady as anemia and tilted uterus. Various treatment was rendered the patient by the surgeon but the pain and suffering persisted. Following the operation she lost about 30 pounds, was unable to eat solid foods, was unable to sleep, vomited frequently, became extremely nervous, and was so physically incapacitated that it was impossible for her to fulfill her duties as a wife and mother.

The patient underwent a second abdominal operation performed by a second surgeon. During the operation there was removed from her large bowel, where it joined the small intestine, a cloth sack approximately 10 inches wide by 16 or 18 inches long. These two operations were the only ones ever performed on her body.

The patient brought an action in the Superior Court of Maricopa County, Arizona, against the surgeon for injuries to her person which she suffered by reason of his alleged negligence in the performance of the first operation. The court entered judgment in favor of the surgeon, and the patient appealed.

The Supreme Court of Arizona reversed the decision of the court below. The Supreme Court declared: "Where the thing which caused the injury complained of is shown to be under the management of defendant (the surgeon) . . . and the accident is such as in the ordinary course of things does not happen if . . . (he who has) its management or control . . . (uses) proper care, it affords reasonable evidence, in the absence of explanation by defendant, that the accident arose from want of care. . . . In other words, when such circumstances are shown to exist, the inference arises that defendant is guilty of negligence, and, in the absence of explanation by defendant, justifies a recovery in damages for such wrong."

McCOWEN VS. SISTERS OF MOST PRECIOUS BLOOD OF ENID

Supreme Court of Oklahoma

253 P. 2d 830

The Sisters Adorers of the Most Precious Blood of Enid, a corporation, was the lessee and operator of the Stillwater Municipal Hospital. The patient was admitted to the hospital for the purpose of an operation by her surgeon. She was prepared and taken to the operating room by nurses employed by the corporation and placed upon an operating table where she was given a "spinal block" by her surgeon.

After administering the anesthetic, the sur-

* Counsellor at Law of the Supreme Court of the United States of America.

geon left the patient in the charge of the nurses nd went into an adjoining room to scrub his ands in preparation for the operation. During is absence, and without any instructions from im but in line of duty, a nurse proceeded to lace the patient's legs in devices commonly eferred to as stirrups. These instruments were ttached on each side of the foot or lower end f the operating table, were held in place by set of screws and could be removed from the able, or raised or lowered to accommodate the ize of the patient. They were designed in such a way that the patient's legs, from the hips o the knees, were held in a vertical position while the calves of the legs were permitted to rest in the trough-like devices and extend in a horizontal position.

When the patient's legs were placed in the stirrups, the lower end of the table was let down by means of a screw type crank which permitted the lower part of her back to extend over the end of the table. When the drop leaf of the table was let down, the entire weight of the lower part of the patient's body was supported by the stirrups. While the patient was in this position, the surgical nurse (a second nurse), observed that the stirrups were improperly attached, or were in reverse order, and suggested that the nurse change them so that they would be more comfortable and not interfere with the patient's circulatory system. Without raising the drop-leaf end of the table, the nurse proceeded to change the stirrups and in doing so, permitted the patient's legs to fall to the floor which caused her body to slide forward over the end of the table. The fall caused severe injuries to the patient's back.

The patient filed an action in the District Court of Payne County, Oklahoma, against the corporation to obtain damages for her injuries. The corporation argued that the nurse, although not in the regular employ of the surgeon, was under his special supervision and control during the operation and, therefore, that the relationship of master and servant existed and the surgeon, rather than the corporation, was liable for the nurse's negligence. The court entered judgment in favor of the corporation, and the patient appealed.

The Supreme Court of Oklahoma reversed the decision of the lower court. The Supreme Court stated: " . . . the preparation of . . . (the) patient for surgery in the hospital operating room . . . (was) done by the hospital's nursing staff and the charges therefor are a part of the hospital fees and not the surgeon's fee."

BOOK REVIEWS

Surgical Treatment of Unequal Extremities. By Charles Weer Goff, M.D. 184 pages. Charles C Thomas. Springfield, Ill., 1960. *$11.00.*

Doctor Goff is already well known for his work on disturbances of growth in children, and has been an instructor on the "Surgical Care of Unequal Extremities" for the American Academy of Orthopaedic Surgery.

In this book, the author gives briefly a comprehensive account of his personal experiences in the surgical treatment of unequal extremities of children at the Newington Hospital for Crippled Children, and gives a résumé of the work done by other authorities in dealing with this problem. The dangers and complications of lengthening a leg by surgery are discussed. The methods of measuring the legs accurately are given with a timely warning about the indiscriminate taking of roentgenograms.

A chapter is devoted to the stimulation of growth in long bones and mentions venous stasis and intentional osteotomy also of the long bones. Foreign substances and the insertion of dissimilar screws near the epiphyseal plates are evaluated and seem to cause no significant growth stimulation. The author was not enthusiastic about the lumbar sympathetic gangliectomy. No mention was made of the effect of an induced arteriovenous fistula.

In Chapter v, growth control by staples, and the permanent epiphysiodesis of Phemister are described. Also, several excellent growth charts are included which will be of use to every clinician interested in this problem.

This carefully written book should be available to every surgeon contemplating the treatment of unequal leg length in children.

B. W.

Atlas of Anatomy and Surgical Approaches in Orthopaedic Surgery—Lower Extremity, Volume 2. By Rudolfo Cosentino, M.D. 264 pages. Charles C Thomas. Springfield, 1961. *$14.00.*

From the title one expects that this is just another book on surgical approaches. This is certainly not the case and the author has contributed a fascinating and exciting textbook, if such there can be, on anatomy and surgical approaches to the extremities. This is done by a series of regional, black and white photographs. The dissection of each region is excellent and the significant structures are individually labeled. The photographs are superb. There is an explanatory legend accompanying each photograph. After the entire region is developed by these series of anatomic studies, one or two of the incisions used to expose this area are presented. In this fashion the author acquaints the reader with how "to visualize accurately what lies just beyond the retractor."

Many of us prefer different types of medical illustrations rather than actual photographs of an anatomic specimen. In this text, however, this prejudice will be cast aside because of the excellence of both the anatomic dissections and the photography. In volume two the regions covered are the lumbar and sacral plexus, the gluteal region and the lower extremities.

This book should supplement other standard textbooks on anatomic exposures. It should be included in most libraries on anatomy as well. It is highly recommended for those physicians who perform extremity surgery or for those who are preparing for their specialty boards or surgical board examinations. The author deserves the highest commendation.

P. R. M.

The Rehabilitation of Patients with Deep Burns Connected with the Recovery of Functions and Sensation of Grafted Skin Areas. A Clinical, Histological and Experimental Study. By A. J. Sneep, J. F. Folkerts and H. A. Meyling. 70 pages. North-Holland Publishing Co. Amsterdam, 1959. *$7.00.*

The object of this book is to consider: (1) local treatment for burns and the recovery of sensation in the affected skin areas and (2) recovery of function of the skin areas of the hands and joints.

The authors carried out primary excision and autografting if the burn was limited to 20 per cent of the body surface. The pin prick test was used to establish full thickness loss. In the more extensive burn they were not impressed with the use of homografts in the late treatment but felt that they were of advantage when the early excision technic was used.

Primary excision and autografting of burns of the hand were carried out early despite the extent of the burn. This greatly shortened the rehabilitation of these hands.

The use of embryonal grafts is discussed.

The authors, in clinical and experimental tests, found that primary excision and application of autografts resulted in almost complete return of sensation and that the graft takes on the sensory pattern of the surrounding skin. The recovery in secondary grafts was less favorable. The extensive neurologic and histologic investigations presented appear to justify these observations.

The Sturge-Weber Syndrome. By G. L. Alexander, M.D., F.R.C.S.E., F.R.C.S. and R. M. Norman, M.D., M.R.C.P. 95 pages. John Wright & Sons Ltd., Bristol, England (Williams & Wilkins Co., American agent), 1960. *$7.50.*

This volume deals with the Sturge-Weber syndrome and is written in the typically precise and dissecting British manner. Great care has been taken to clearly define and delineate the syndrome as it was originally described by Sturge in 1879.

Seven cases are presented in detail and include case histories, psychologic testing, arteriography, electroencephalography, operative findings, electrocorticography, convalescent period and follow-up.

The radiologic aspects of the Sturge-Weber syndrome are dealt with in a separate chapter and include descriptions of skiograms as well as arteriograms.

An excellent section is then offered presenting a detailed and painstaking description of the electroencephalographic and electrocorticographic change in these cases.

Interesting concepts and descriptions of neuropathologic findings in resected specimens are well presented with accompanying illustrations and photomicrographs.

Theories on the embryonal causation and origin of the facial nevus and cerebral angiomatosis are related in an interesting manner. Later sections dealing with the epileptic manifestations and ophthalmologic aspects are clearly reviewed.

This monograph is to be highly recommended to both the student of neurology and the student of neurological surgery.

J. M.

Medical, Surgical and Gynecological Complications of Pregnancy. By the staff of the Mount Sinai Hospital. Edited by Alan F. Guttmacher, M.D. and Joseph J. Rovinsky, M.D. 619 pages. Williams & Wilkins Co. Baltimore, 1960. *$16.50.*

The medical profession and the obstetricians and gynecologists in particular have had at their disposal for many years good obstetric texts and references. Now with this volume there exists one of the most valuable supplements to the field that has been put into print in many years. Most standard books adequately cover normal physiology and the mechanics of obstetrics but the complications of pregnancy necessarily suffer if for no other reason than the lack of space in a single volume. This new work magnificently covers all of the medical, surgical and gynecologic complications that may occur during pregnancy. Following each chapter is an unusually adequate and complete list of references that includes, for the most part, only material published since 1950. The contributors are all men well qualified to write and teach in their field. Each man presents his material clearly and completely. No one in the field of obstetrics, whether as a member of a teaching staff or as a practitioner, should fail to read this book or fail to have it available for frequent reference.

J. C. U.

Instructional Course Lectures. Volume 17. The American Academy of Orthopedic Surgeons. Edited by Fred C. Reynolds, M.D. 421 pages. C. V. Mosby Co. St. Louis, 1960. *$18.50.*

The seventeenth volume of selected course lectures edited by Dr. Reynolds deals with the general topics of fractures, bone graft surgery, children's orthopedics, athletic injuries and disability evaluation. The lectures are printed in the well edited form consistent throughout this series.

The limitation to 500 reprints by contributors will undoubtedly stimulate the value of this volume. It is an excellent reference book for the practicing orthopedist and it gives a concise and comprehensive review of the subjects covered. The orthopedic trainee will enjoy the bold printing and generous number of illustrations which make it easily read and assimilated.

The sections dealing with bone growth in children as well as the comprehensive discus-

sion of bone graft surgery deserve a special commendation.

R. F. S.

The Operations for Inguinal Hernia. Contributions of Bassini, Halsted, Andrews, Ferguson, and Lotheissen, and a Current Recommendation. By Mark M. Ravitch, M.D. and James M. Hitzrot, M.D. 64 pages. C. V. Mosby Co. St. Louis, 1960. *$4.00.*

This booklet on inguinal hernia will make an interesting and informative addition to the surgeon's library. In it the authors review the original papers of Bassini, Halsted, Andrews, Ferguson and Lotheissen on repair of the inguinal hernia. Quotations and plates from the original articles contribute added interest and color to the text. The material is well organized and easy to read. A clear picture is given of the fundamental differences between the various types of repairs and their development is presented in the proper historical perspective. At the end of the review the current technic employed in the "Johns Hopkins hernia repair" is described in detail.

A. R. B., Jr., R. McP. and F. M.

Medical-Surgical Nursing. Second Edition. By Kathleen Newton Shafer, Janet R. Sawyer, Audrey M. McCluskey and Edna Lifgren Beck. 876 pages. C. V. Mosby Co. St. Louis, 1961. *$8.75.*

The second edition of this book has been changed somewhat in general format. The two column pages and the addition of many more subheadings should make this book easier to read. New content and more comprehensive discussions greatly enhance its value. Clear photographs, diagrams and meaningful tables supplement the text.

The authors have emphasized the importance of nursing care of patients who have diseases requiring medical and/or surgical treatment. This is a condensed, well written text. It should be useful in teaching nursing care of patients undergoing either medical and/or surgical treatment whether the two subjects are taught together or separately. This book also serves as a valuable reference for graduate nurses.

H. L. W.

Rose and Carless' Manual of Surgery. Nineteenth Edition. Edited by Sir Cecil Wakeley, Michael Harmer and Selwyn Taylor. 1,389 pages. Williams & Wilkins Co. Baltimore, 1960. *$15.00.*

The subject matter in this text is arranged in seven sections including equal space for orthopedic, neurologic, urologic and alimentary tract surgery. A section on head and neck surgery also includes sections on ear, nose, throat and ophthalmology. While the scope of this book is wide, detail is necessarily limited.

Included in this volume are 370 new illustrations and twelve color plates. This book serves as a ready reference because of the wide range of subject matter under one cover.

W. P. and R. McP.